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Drinking Water Criteria Document for Uranium

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16. ABSTRACT

The Office of Drinking Water (ODW), U.S. Environmental Protection Agency has prepared a Drinking Water Criteria Document on Uranium. This Criteria Document is an extensive review of the following topics:

- Physical and chemical properties of Uranium
- Toxicokinetics and human exposure to Uranium
- Health Effects of Uranium in humans and animals
- Mechanisms of toxicity of Uranium
- Quantification of toxicological effects of Uranium

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I. SUMMARY

This Criteria Document discusses and analyzes the existing information for analysis, treatment, occurrence, relative source contribution and health effects of uranium in drinking water. Uranium is both chemically toxic to the kidney and radiotoxic to bone and bone marrow. The AADI for uranium in drinking water is in the range of 4 to 40 pCi/l. A Maximum Contaminant Level (MCL) has already been promulgated for radium (5 pCi/L), and since it is also a bone seeker, the health effects and risk of uranium due to its radiotoxicity can be compared to those of radium. Using this basis the standard for uranium in drinking water based on radiotoxicity could be in the range of 10 to 40 pCi/l. The effects of consuming water containing 10 and 5 pCi/L of uranium and radium, respectively, have not been directly demonstrated for ingestion at these environmental levels. Due to the variation in background cancer cases, the number of people needed to demonstrate the effect is prohibitively large. Between 100 and 2,000 public water supplies in the United States (out of a total of 63,975) exceed 10 pCi uranium/L. Most of the uranium ingested by humans comes from drinking water.

II. GENERAL INFORMATION AND PROPERTIES

Uranium, the element with the highest atomic number of any naturally occurring element (unless one counts the "natural" fission reactor at Oklo Gabon, Africa, as a natural event (Cowan 1976)), is found in the earth's crust and waters. Its concentration in a few ores is in the range of 40% to 60%. About 100 mineral species contain 1% or more, and the average concentration of uranium in the earth's crust is 4 x 10^{-4}% (Hursh and Spoor 1973). It is found in rocks such as lignite, monazite sands and phosphate rocks, as well as in minerals such as carnotite and pitchblende (CRPB 1980). The concentration in phosphate rocks can be as high as 0.12 mg/g. Seawater concentrations of uranium range from 0.00015 to 0.003 mg/L. Uranium is also found in minerals, such as apatite, sphene and zircon, which are found in granites and metamorphic rocks. All of these sources can come in contact with water which may be used for drinking purposes. Thus, it is expected that some drinking water sources will contain measurable concentrations of uranium.

Uranium exists in valence states +2, +3, +4, +5 or +6, with the most common being the hexavalent state. There are 15 isotopes of uranium, three of which occur in nature (see Table II-1). The hexavalent state is the important one in water, since almost all tetravalent compounds are practically insoluble. In nature, uranium is commonly found in the uranyl

Table II-1 Properties of Naturally Occurring Uranium

Isotope	% in Natural uranium	Half-life (years)	Alpha energies in MeV (abundance)
^{234}U	0.006	2.47×10^5	4.77 (72%) 4.72 (28%)
^{235}U	0.72	7.1×10^8	4.58 (8%) 4.40 (57%) 4.37 (18%)
^{238}U	99.27	4.51×10^9	4.20 (75%) 4.15 (25%)

To relate mass and activity, use the relation:

$$\frac{dN}{dt} = \lambda N$$

where N is the number of radioactive atoms, and is the decay constant ($= 0.693/T_{1/2}$, where $T_{1/2}$ is the half-life). Then, 1 g of uranium has the following activity:

$$1 \text{ g of U} = \frac{(0.693)\,(6.023 \times 10^{23})}{(3.15 \times 10^7)(3.7 \times 10^{10})} \times \frac{P_i}{A_i (T_{1/2})_i} = \text{Ci of U}$$

where 3.15×10^7 sec = 1 year; $(T_{1/2})_i$ is the half-life in years for the i^{th} isotope; Avogadro's number, 6.023×10^{23}, is the number of molecules in moles; A_i is the atomic weight; 3.7×10^{10} is the number of disintegrations/sec in 1 Ci and P_i is is the percentage of the i^{th} isotope in naturally occurring uranium.

Table II-1 - (continued)

Then:

$$1 \text{ g of U} = \frac{(0.693)(6.023 \times 10^{23})}{(3.15 \times 10^{7})(3.7 \times 10^{10})} \times \left[\frac{6 \times 10^{-5}}{(234)(2.47 \times 10^{5})} + \frac{7.2 \times 10^{-3}}{(235)(7.1 \times 10^{8})} + \frac{0.9927}{(238)(4.51 \times 10^{9})} \right] \text{Ci}$$

$$= 0.72 \times 10^{-6} \text{ Ci}$$
$$= 0.72 \text{ microcuries}$$

Thus,

1 microgram of U_{Nat} = 0.72 pCi of U_{Nat}

or

10 pCi of U_{Nat} = 14 micrograms of U

ion, UO_2^{2+}. All natural compounds of uranium contain oxygen (Katz and Rabinowitch 1951).

The best known modern use of uranium is as a fuel in nuclear power reactors. It is also used in inertial guidance devices and gyro compasses, as a counterweight for missile reentry vehicles, as shielding material and as x-ray targets. Uranium compounds are used in photographic toners, colored glass, ceramic glazes, tiles and dental porcelain and as catalysts. Some of these uses predate the nuclear age.

Uranium may exist in one of three allotropic forms: orthorhombic, tetragonal or body-centered cubic. As a metal, it is a strong reducing agent. Uranium is not very stable in the 3+ valence state (U^{3+}), and in the 4+ valence state (U^{4+}), it oxidizes slowly in air. In the 5+ valence state (UO_2^+), it is only stable in the pH range of 2 to 4; it is most stable in the 6+ valence state (UO_2^{2+}). The hexavalent state can be produced by dissolving uranyl salts, such as halide, nitrate, sulfate or acetate. There is no evidence to suggest that uranyl is essential to man (Hursh and Spoor 1973).

Hydrolysis plays an important role in the environmental behavior of uranium - it competes with organic and inorganic moieties, with the formation of complexes. At pH 6 and below, U forms very stable complexes with organic compounds that are rich in oxygen in the form of humic and fulvic acids in the natural environment. This interaction involves peats, coals and asphalts, as well as inorganic interactions with shales, phosphorites and carbonates (Katz and Rabinowitch 1951).

As shown in Table II-1, the relationship between mass and activity depends on the isotopic composition. The calculation shown uses the steady state equilibrium values.

Uranium, like some other elements, can be tasted at some levels in drinking water. A preliminary taste test by a panel of 30 people revealed that 5 ppm of soluble, natural uranium represented the threshold of taste (Public Health Service 1965). This was once used as the criterium for a standard.

A. Analysis of Uranium in Drinking Water

The regulations for radioactivity in drinking water (41 FR 28402, 1976) list the American Society For Testing and Materials (ASTM) D-2907 Method (see Appendix II), which is a fluorometric method, for analyzing uranium. If the standard for uranium were to be any less than 50 micrograms/L (about 33 pCi/L), this method would be unacceptable, since its sensitivity limit is 5 micrograms/L. This is because the sensitivity limit must be 1/10 the standard or lower. The ASTM method does have several possible interferences.

The EPA fluorometric method (see Appendix III) has a lower limit of detection or sensitivity of 0.1 micrograms/L and has no apparent interferences. This is the preferred method.

The most reliable and cost-effective method for determining the concentration of uranium in drinking water is the fluorometric method (Kreiger and Whittaker 1980), which is being considered as the nationally accepted one. This method measures the mass of uranium present (essentially, the amount of ^{238}U present, since it represents 99% of the mass). If the uranium isotopes are in equilibrium and the ratios are those that occur naturally, then this mass number can be converted to activity. However, the $^{234}U/^{238}U$ activity ratio is often greater than its equilibrium value of 1; in the U.S., it can be as high as 16 or more, although most ratios are lower than 3 (for example, see Table IV-6). To determine the range of concentrations that could represent a problem area, assume that the $^{234}U/^{238}U$ activity ratio is 3. Then, if the measured value is 15 micrograms/L, the normal conversion (shown in Table II-1) would yield 10 pCi/L. However, due to the extra activity, the actual value is about 20 pCi/L. At the other extreme, if the measured value is 7.5 micrograms/L, it would convert the equilibrium to 5 pCi/L. Due to the extra activity, the actual value is about 10 pCi/L. Thus, if the measured value exceeds 15 micrograms/L, the Recommended Maximum Contaminant Level (RMCL) of 10 pCi/L would be exceeded. Also, if the measured value is less than

7.5 micrograms/L, it is reasonably clear that the RMCL would not be challenged. For measurements in this range, either the $^{234}U/^{238}U$ ratio or the radioactivity should be required to be measured. This can be done by measuring the alpha particle spectra, possibly with a surface barrier detector and multi-channel analyzer. The direct method (see Appendix III) described by Kreiger and Whittaker (1980) does not measure the separate isotopes, but it does determine the radioactivity level.

An estimate of the potential impact of assuming isotopic equilibrium or using only the fluorometric method can be determined by estimating how many public water supplies (PWSs) have uranium concentrations between 10 and 20 pCi/L. From an Oak Ridge National Laboratory (ORNL) report (Drury et al. 1981), between 64 and 1,920 PWSs exceed 10 pCi/L, and between 26 and 640 exceed 20 pCi/L. Thus, about 1,000 PWSs have concentrations between 10 and 20 pCi/L. Assume that about 20% of these have $^{234}U/^{238}U$ activity ratios that exceed 2. Then, 200 supplies would be missed if only the fluorometric method were used. At a level of 4×10^{-5} excess deaths/lifetime and an average population of 2,000/PWS, the cost in lives/lifetime would be:

$$\frac{4 \times 10^{-5} \text{ excess deaths}}{\text{lifetime}} \times \frac{(2{,}000 \text{ persons})}{\text{PWS}} \times 200 \text{ PWSs} = 16$$

where 4×10^{-5} is the number of excess deaths/lifetime for ingestion of water with a concentration of 10 pCi/L. Although admittedly crude, this approximation gives some idea of the impact of using only the fluorometric method for determining uranium concentrations in drinking water. This estimate can be used in balancing the cost of the extra radiological monitoring against the health risk.

The uranium concentration in groundwater can vary with time. It has been shown to vary by as much as an order of magnitude over the period of a year (Savannah River 1979). Thus, the samples for analysis should be collected at least quarterly.

B. Transport

The general population is exposed to uranium primarily through food and drinking water, ingesting approximately 1 to 2 micrograms/day via food. The uranium is ingested as soluble uranyl salt or complex or is oxidized in the body to uranyl.

The two transportable (soluble) forms of uranium that are most likely to be ingested and found in the human body are the hexavalent and tetravalent forms. The rate at which uranium is absorbed, retained or excreted from the GI tract or the lung is dependent on its solubility and other factors such as particle size, surface characteristics, temperature and pH, also affect these rates. Table II-2 lists the solubility (sometimes called transportability) of several uranium compounds. Uranium can

Table II-2 Classification of Uranium Compounds in Water According to Transportability in the Body[a]

Highly transportable	Moderately transportable	Slightly transportable
UF_6	UO_2	UO_2
UO_3	U_3O_8	U_3O_8
$UO_2(NO_3)_2$	UO_3	Uranium oxides
UF_4	UF_4	Uranium hydrides
Uranium sulfates	Uranium nitrates	Uranium carbides
Uranium carbonates		Salvage ash

[a] The transportability of UO_3, UO_2 and U_3O_8 depends on the temperature. As the temperature goes up, the transportability decreases.

From Berlin and Rudell (1977).

be organically bound, e.g. in food, but little is known about this organic bonding.

The hexavalent and tetravalent forms can be changed into each other by oxidation or reduction in the body. Since the gut is essentially a reducing environment, the tetravalent form could predominate there. Once in the body, the soluble uranium complexes with anions and bases such as bicarbonates, citrate, malate and lactate, primarily reacting with protein and bicarbonates (Berlin and Rudell 1977). The uranyl compounds interact strongly with phosphate-containing molecules and tissues and with carboxyl and/or hydroxyl groups such as proteins, nucleotides and bone tissue.

In the mammalian organism, uranium tends to be converted into the water-soluble hexavalent form. Uranyl fluoride (UO_2F_2) and uranyl nitrate ($UO_2(NO_3)_2$) are very soluble in blood plasma (Adams and Spoor 1974). In the blood, 47% of the uranium is found complexed with the bicarbonate in plasma, 32% is bound to plasma proteins, and 20% is bound to erythrocytes (Berlin and Rudell 1977). The fraction that is bound with the bicarbonate is ultra-filterable. This is taken up by bone or is filtered by the glomeruli into the urine. In this manner, the plasma is depleted of hexavalent uranium. More hexavalent uranium then returns to circulation from the extracellular fluid.

Thus, the fraction complexed by protein is continually shifted to an ultra-filterable form until all of it is cleared from the blood.

III. TOXICOKINETICS

A. Absorption

The primary chemical toxic effect of uranium is on the kidney. Thus, to produce toxicity, ingested uranium must be absorbed from the digestive tract into the bloodstream. The degree of absorption that will occur in a given situation depends on several factors, including the solubility of the uranium compounds involved, the presence or absence of food in the digestive tract, age, whether the uranium dose is acute or chronic, etc. The extent of gastrointestinal absorption of uranium may be determined by experimental administration or of actinide or by measuring dietary intake and in both cases measuring fecal and urinary excretion.

Wrenn et al. (in press) have thoroughly reviewed the experimental studies of absorption of ingested uranium. These studies date from the 1940s to the present. For example, Hamilton (1948) administered uranyl nitrate to 60-day-old rats by gavage at a dosage of 0.3 mg U/kg and reported an absorption of less than 0.05%. These results were later recalculated by Durbin and Wrenn (1975) to an absorption of up to 0.35%. A study of 10 dogs ingesting a single dose of uranyl fluoride yielded an average absorption factor of 1.55% where the doses were 0.007 and 0.7 mg/kg (Fish et al. 1960). Sullivan (1980) gavaged rats with either ^{232}U or ^{233}U nitrate and reported absorption values of 0.06% for both isotopes. Harrison and Stather (1981) gave gavage doses of 0.63 mg U/kg to adult Syrian hamsters and calculated absorption to be 0.77%. Larsen et al. (1984) gavaged a single adult baboon with a dose of 0.53 microgram U/kg and calculated a 1.2% absorption. There is, however, a wide variation among species and between animals and humans.

Only very limited data exist for uranium absorption by humans following oral administration. Hursh and Spoor (1973) have reviewed these results. In one study, a single human volunteer drank an aqueous solution containing 1 g uranyl nitrate (Butterworth 1955). Unfortunately, he quickly became ill, vomiting up a portion of the dose. Based on subsequent excretion data, it was estimated that he had absorbed about 1% of the dose. Hursh et al. (1969) gave uranyl nitrate doses of from 0.08 to 0.17 mg U/kg in Coca Cola to four hospital patients. Based on subsequent measurements of uranium excreted in urine, a mean absorption of 1.4% was determined.

The above studies involved only a single administration of uranium. Continuous or chronic administration is, of course, more relevant to public health considerations, but the number of such studies is limited. Tracy et al. (1983)

administered uranyl nitrate in drinking water to rabbits and rats for 90 days and calculated the resultant absorption of uranium at 0.28% and 0.035%, respectively.

Other chronic studies have involved the feeding of uranium salts in the diet. Maynard et al. (1953) fed rats for up to two years on diets containing from 0.05% to 2.0% uranium salts and found that absorption ranged from 0.038% to 0.078%. The percent absorption appeared to be independent of the dietary concentration. Haven and Hodge (1949) gave rats uranium in their food for 30 days at doses ranging up to 12%. The absorption results were comparable to those of Maynard et al.

Table III-1 summarizes the results of the major experimental studies of uranium absorption in animals and man following oral administration of either single or chronic doses. Sullivan's high absorption values in the rat were obtained by supplementing ^{233}U nitrate with oxident fereic nitrate (Sullivan, 1984).

No experimental studies of chronic uranium uptake by humans have been performed. However, absorption can be roughly estimated by comparing daily dietary intake and daily excretion of uranium. Alternatively, one can compare standard dietary intake values with actual tissue concentrations of uranium determined on autopsy samples. Accurate determinations of such values for sufficiently large numbers of people are obviously quite difficult to secure.

Values derived from such environmental data vary widely. For example, the dietary and tissue concentrations of uranium found by Welford and Baird (1967) in individuals in three U.S. cities suggested absorption values as high as 31%. This study assumed that the individuals whose tissues were sampled had consumed an "average" diet for their city with regard to uranium content -- a questionable assumption. By contrast, a concurrent study of dietary intake and urinary excretion in three Japanese villages suggested uranium absorption values of only 1.6% (Yamamoto et al. 1968, Masuda 1971a,b,c,d). A very thorough study, but of only one individual, in an area of India with elevated levels of uranium in the drinking water indicated a G.I. absorption value of 2.2% (Somayajulu et al. 1980). These latter two values are in agreement with the experimental studies previously cited.

Table III-1 Results of Experimental Studies of Gastrointestinal Absorption of Uranium in Adult Animals and Humans

Species	Uranium absorption (%)	Uranium uptake (micrograms U/kg/day)	References
1. Single administration			
Hamster	0.77	6.3×10^2	Harrison and Stather (1981)
Dog	1.55	7×10^2	Fish et al. (1960)
Baboon	1.2	5×10^{-1}	Larsen et al. (1984)
Man	0.73	6.7×10^3	Butterworth (1958)
	1.4	1.32×10^2	Hursh et al. (1969)
Rat	0.35	3×10^2	Hamilton (1948)
	0.06	2.3	Sullivan (1980a)
	0.06	4×10^3	
	0.044	5.1×10^3	Sullivan (1983)
	0.044	1.3×10^4	
	0.088	2.5×10^4	
	1.59	30	La Touche and Willis (1984)
	1.55	3×10^2	
	1.68	3×10^3	
	0.56	3×10^4	

Table III-1 - continued

Species	Uranium absorption (%)	Uranium uptake (microgram U/kg/day)	References
Rat			
fed	0.07	5.1×10^3	Sullivan (1984)
fasted	0.17	5.1×10^3	
fasted+Fe^0	0.13	5.1×10^3	
fasted+Fe^{3+}	3.26	5.1×10^3	
2. Chronic administration			
Rabbit	0.28	17×10^3	Tracy et al. (1983)
Rat	0.035	3.3×10^4	
	0.052	2×10^4	Maynard et al. (1953)
	0.059	9.6×10^4	
	0.06	2×10^5	
	0.038	1.2×10^5	
	0.078	4.7×10^5	
	0.04	9.7×10^5	Haven and Hodge (1949)

Adapted from Wrenn et al. (1985).

Wrenn et al. (1985) have recently made a thorough review of the literature in this field. They came to the conclusion that a G.I. absorption of 1.4% at environmental levels of uranium uptake was the best current estimate. Furthermore, they felt that this value might even decrease slightly with increasing uranium intake. Thus, for a regulatory model, it seems preferable to use an absorption of 1.5% to 2.0%, even though this is lower than the value specified in ICRP 30 (1979).

Absorption may be modified by several factors. For example, age-dependent absorption has been demonstrated in animals (Sullivan 1980b, 1982,1983). Newborn rodents, swine and sheep transport quantities of radionuclides across the intestine that are orders of magnitude higher than those for adults. For the actinide elements (uranium, plutonium, neptunium, americium, californium and einsteinium), absorption in neonatal rodents is usually about two orders of magnitude higher than in adult rodents. This effect is thought to be due to the enhanced ability of the young to absorb intact proteins. This enhanced permeability varies among species of mammals such as rats, guinea pigs, dogs, swine and humans.

For single doses of uranium gavaged to rats, the percentages retained are seen in Table III-2 (Sullivan 1980a,b). As shown, the neonatal rats absorb about 20 times as much uranium nitrate as the adult rats and about 10 times as much uranium

Table III-2 Retention of Uranium by Rats as a Function of Age

Uranium dose (micrograms)	% Uranyl nitrate retained		% Uranium oxide retained	
	Neonatal	Adult	Neonatal	Adult
.1167	6.7 ± .98	.061 ± .008	---	---
211	1.3 ± .50	.061 ± .008	.359 ± .065	.037 ± .007

From Sullivan (1980a,b).

oxide as the adults. Similar results have been reported elsewhere (Bulman 1980). Newborn swine absorb more than 200 times more than adult swine (Sullivan, 1982, 1983).

The skeletal content of elements such as U, Ra and Pb generally increases rapidly from conception and reaches an equilibrium in adult years. It appears, however, that the dose to mass ratio for bone is somewhat independent of age (Wrenn 1977). Although no direct evidence of this exists for uranium, it appears to be true for ^{90}Sr (Kahn et al. 1969) and radium (Wrenn 1977). A number of factors can combine to bring this about; important among these is the fact that children consume different amounts of food and water than adults and are growing in size. Although there is no direct evidence that the uranium dose to bone is independent of age, the evidence for a similar bone seeker, radium, is suggestive of this relationship.

Although there is no information about human absorption of uranium with respect to age, it is possible that it will be similar to the above findings for animals. The results to date, however, do not appear quantitative enough to consider in the determination of a standard.

A related matter is the extent of placental transfer of uranium from the mother to the fetus. Sikov and Mahlum (1968) studied this in rats using uranium, neptunium and plutonium. They found that the placenta and fetal membranes were distinct barriers to the passage of these actinides from the maternal circulation to the fetus. Thus, it appears that no special concern need be given to this matter in setting drinking water standards in the absence of contradictory human data.

B. Distribution

Uranium absorbed from the digestive tract quickly leaves the blood. La Touche and Willis (1984) gavaged adult rats with uranyl nitrate solutions at four different doses and followed the time course of distribution in various tissues from 15 minutes to 10 days. Table III-3 shows their data for a gavage dose of 0.3 mg U/kg. Blood clearance of injected uranium is also rapid. Using beagle dogs, Stevens et al. (1980) found that up to 60% of an injected dose of uranium was excreted within 24 hours. In humans, 99% of injected uranium has left the blood after 24 hours (Luessenhop et al. 1958).

The distribution of this absorbed uranium into organs and tissues has been studied extensively in animals following administration by injection, inhalation and ingestion (Yuile 1973, Hursh and Spoor 1973, Durbin and Wrenn 1975, Stevens et al. 1980, Sullivan 1980a,b, Sullivan and Gorham 1982, Harrison and Stather 1981). For adult rats, the pattern of distribution

Table III-3 Uranium Concentrations in Rat Tissues at Various Times Following Oral Administration of Uranyl Nitrate Solution[a]

	Uranium concentration, ng/g dry wt. (mean ± S.E.)				
Time (hr)	Kidney	Femur	Blood	Liver	No. Animals
0.25	69 ± 16	13 ± 2	7 ± 2	5 ± 2	8
0.5	108 ± 32	12 ± 2	8 ± 2	3 ± 1	8
1.0	147 ± 26	21 ± 4	3 ± 2	2 ± 1	8
2.0	438 ± 84	32 ± 6	3 ± 2	3 ± 1	7
4.0	469 ± 62	36 ± 4	6 ± 1	6 ± 1	8
8.0	685 ± 68	48 ± 3	5 ± 3	4 ± 1	7
24.0	446 ± 98	30 ± 5	3 ± 1	5 ± 1	8
48.0	142 ± 16	17 ± 3	3 ± 1	3 ± 1	8
96.0	247 ± 35	37 ± 5	4 ± 1	4 ± <1	8
240.0	77 ± 10	24 ± 4	3 ± 1	2 ± <1	6

[a] 0.3 mg/kg as uranium. Male Wistar rats, averaging 200 g, were used. Minimum detection limit was 2 ng.

Adapted from La Touche and Willis (1984).

following injection is 5% to 20% to the skeleton, about 20% to the kidney and 50% to 75% excreted in urine in the first 24 hours (Adams and Spoor 1974). For humans, the distribution is approximately 6% to 24% to the skeleton, about 16% to the kidney and 48% to 49% excreted in urine in the first 24 hours (Luessenhop et al. 1958). Thus, bone and kidney are the major tissues of uranium accumulation. Other soft tissues attain much lower concentrations.

Animal studies of oral administration of soluble uranium compounds confirm this preferential uptake by kidney and femur (Hursh and Spoor 1973). Uranyl nitrate and UO_2F_2 produced uptakes of 20 to 30 micrograms/g (fresh weight) in the kidney, while UO_2F_2 at 0.5% in the diet produced an uptake of 60 micrograms/g (ash) in the femur, and uranyl nitrate at 2% in the diet caused 150 to 200 micrograms/g (ash) in the femur. At 0.5% uranyl nitrate in the diet, the amount deposited in the bone was roughly equal to that excreted, or about 2.4 micrograms/day. At 1% uranyl nitrate in the diet, about 0.1% of the soluble uranium compound was absorbed by the body.

Continuous ingestion or inhalation of uranium in animals results in directly proportional amounts in bone and soft tissues. In fact, equilibrium concentrations of uranium are reached rather quickly in all tissues of growing animals and in the soft tissues of mature animals (Wrenn et al., 1985). It appears that nearly constant uranium concentrations should be present in the tissues of humans exposed to a steady level of environmental uranium. Furthermore, uranium turnover in soft tissues is fairly rapid, while turnover of at least some fraction in skeleton is slow.

Uranium in the kidney first becomes deposited along the surfaces of the proximal convoluted tubules of the outer and mid-zones of the cortex. The uranium is concentrated in the cortical zone of the kidney (Luessenhop et al. 1958), and the uranyl bicarbonate complexes pass through renal glomeruli. As the pH of the glomerular filtrate decreases and the bicarbonate becomes absorbed by the cells of the proximal convoluted tubule, the uranyl cations are liberated to combine with the protein along the tubular walls.

In bone, the uranium goes mainly to the periosteal and endosteal bone, with some going to the Haversian System (Luessenhop et al. 1958). The uranium deposition appears to be restricted to the endosteal surfaces and, in particular, to the surfaces of the trabecular bone and the walls of open Haversian canals in the cortical bone (Hamilton 1971). Uranium is initially deposited on the bone surface, but then can redistribute or diffuse into the volume. Both the surface and volume deposits can be quite heterogeneous. The redistribution pattern of ^{233}U from surface to volume is intermediate between that of ^{239}Pu, where most stays on the surface, and that of ^{226}Ra, where most goes to the volume (Stevens et al. 1980).

Cross-placental transfer of uranium citrate has been demonstrated (Adams and Spoor 1974), and thus, it is likely that uranium can accumulate in the bone before birth.

There have been many attempts to establish the "normal" amounts of uranium in the human skeleton. Wrenn et al. (in press) have reviewed these attempts in light of the varied analytical techniques used and the data available as to the human sources. They found that the reported results range from 2.3 to 61.6 micrograms U in the standard Reference Man (see Table III-4). They feel that the best value at present is the arithmetic mean of these, or 24.9 ± 22 micrograms U.

There is even less information on the distribution and amount of uranium in the soft tissues of humans. Wrenn et al. (in press) have reviewed the available data and suggested "best values," i.e., the arithmetic means of the reported mean values (Table III-5). These results require some explanation. First, the very high values noted for lungs and tracheobronchial lymph nodes (TBLN) suggest that much of this uranium resulted from inhalation, not ingestion. Second, the high value for fat is based on only two specimens and seems totally incompatible with animal data. Thus, the total estimated body burden of uranium in soft tissues indicated, 13 micrograms, excludes these three tissues.

Table III-4 - (continued)

Source of samples	Skeletal parts measured	Total U in reference man skeleton (micrograms)	References
India	(Not specified)	13.7	Ganguly (1970)
Nepal	Vertebral bodies	44.8	Fisenne et al. (1983)
Australia	Vertebral bodies and ribs	13.7	Fisenne et al. (1983)

Adapted from Wrenn et al. (1985).

Table III-5 Uranium in Soft Tissues of Individuals With No Known Occupational Exposures

Tissues	Reported uranium concentrations (ng U/g wet tissue)	References	Best values[a]
Liver	0.25	Hamilton (1972)	
	0.13	Welford et al. (1976)	
	0.30	Singh et al. (1983)	0.23
Lung	1.0	Welford and Baird (1967)	
	0.53	Welford et al. (1976)	
	1.7	Singh et al. (1983)	1.08
Kidney	0.24	Welford et al. (1976)	
	0.63	Singh et al. (1983)	0.44
Whole blood	0.72	Hamilton (1970)	
	0.57	Welford et al. (1976)	
	0.11	Lucas and Markun (1970)	0.46
Muscle	0.19	Hamilton (1972)	0.19
Heart	0.16	Hamilton (1972)	0.16
Spleen	0.42	Singh et al. (1983)	0.42
Gonad	0.60	Singh et al. (1983)	0.60
Tracheobronchial Lymph Nodes	24.8	Singh et al. (1983)	24.8
Fat	0.60	Hamilton (1972)	0.60

[a] Best value is the arithmetic mean of the reported values.

Adapted from Wrenn et al, 1985.

One study of uranium content in human tissue for populations in two different Russian towns (Novikov and Abramova 1969) is of particular pertinence. One town had water with elevated uranium concentrations in the range of 40 to 50 micrograms/L, while the other town was more normal. There appeared to be no significant differences between the tissue contents of people from the two towns (See Table III-6). Only 31 individuals were assayed out of a population of about 30,000, however, so these results are not clear-cut.

The fraction of absorbed uranium going from blood to bone (f_2) in man has been determined experimentally as 0.11 to 0.20 (Bernard 1958, Adams and Spoor 1974) and estimated from skeletal burdens assayed at autopsy as 0.17 (Lipsztein 1981). Animal data all suggest values of 0.10 to 0.20 for this fractional transfer (Wrenn et al., 1985). Thus, it appears reasonable to accept a value of 0.20 for regulatory purposes.

The fraction of absorbed uranium going from blood to kidney (f_2) in man is somewhat less than that for bone. Adams and Spoor (1974) suggested a value of 0.12, while Wrenn et al. 1985) proposed 0.11. For regulatory purposes, 0.12 seems appropriate.

Table III-6 Uranium Concentrations[a] in Various Human Tissues[b]

	Town A		Town B
Tissue	Long-term residents (8-63 years), aged 23-74 years; 15 observations	Residents in town for 2-5 years, aged 30-62 years; 7 observations	Long-term residents (14-62 years), aged 24-62 years; 9 observations
Kidney	6.6 ± 1.3	5.1 ± 1.5	4.4 ± 1.4
Sternum	3.0 ± 0.4	3.2 ± 0.9	1.7 ± 0.8
Rib	3.3 ± 0.6	2.8 ± 0.9	2.2 ± 0.8
Vertebra	4.9 ± 0.7	3.2 ± 1.0	2.6 ± 0.8
Liver	2.1 ± 0.9	1.2 ± 0.3	2.5 ± 0.8
Spleen	1.9 ± 0.4	1.9 ± 0.9	1.4 ± 0.5
Brain	1.2 ± 0.5	0.9 ± 0.3	1.1 ± 0.3
Muscle	1.1 ± 0.4	0.9 ± 0.4	0.5 ± 0.2
Blood	0.8 ± 0.3	0.6 ± 0.4	0.5 ± 0.2

[a] Mean ± S.E. The data listed show that the concentration of uranium in the kidneys of persons who had resided in town A was somewhat higher than in town B, but the difference was not statistically significant. A similar pattern was observed in relation to concentrations in sternum, rib and vertebra.

[b] 10^{-9}g Uranium/g raw substance.

From Novikov and Abramova (1969).

C. Elimination

According to Hursh and Spoor (1973), only about 0.1% of uranium ingested from food and water is ultimately incorporated in the human body. We have already seen that only about 1.4% of ingested material is absorbed from the digestive tract. This means that 98% to 99% of the ingested uranium passes on through the digestive tract to the feces, and 90+% of that absorbed is excreted. More than 90% of hexavalent soluble uranium injected intravenously is excreted by the kidney and less than 1% by feces (Canadian Radiation Protection Bureau 1980).

Extensive studies of uranium excretion (retention) have been carried out with various animal species, including beagle dogs (Stevens et al. 1980), baboons (Lipsztein 1981), swine (Sullivan et al. 1982) and, of course, rats (Yuile 1973). Bernard (1958) found that 69% of the hexavalent uranium injected into humans was excreted in the first day, and that 18.5% of the tetravalent uranium was excreted in the first day.

Hexavalent uranium is excreted rapidly by the kidneys. If the pH around the tubules of the kidneys is high, some uranium will be retained there. At low pH, the carbonate uranyl complex is split, with the uranyl ion combining with proteins on the walls and impairing the tubular function (Canadian Radiation Protection Bureau 1980).

There is a fast and a slow phase of excretion, with the latter taking months. The retention half-times of uranium in both bone and kidney are of greatest concern. For bone, half-times of 883 days (Stevens et al. 1980), 180 to 360 days (Hursh and Spoor 1973) and 800 days (Bernard 1958) have been reported. From these and other data, the ICRP 30 (1979) selected half-lives of 20 and 5,000 days as appropriate for the fast and slow components, respectively, Wrenn et al. (1985) prefer 300 days for the fast component.

Retention half-times for uranium in human kidney have been reported as 30 days (Bernard 1958) and more recently as 6 days and 1,500 days, respectively, for the fast and slow components (ICRP 1979). Hursh and Spoor (1973), from measured values after intravenous injection of uranium, prefer a 15-day half-time. This value is incorporated in the model developed by Wrenn et al. (1985).

Although not a lot of information exists for humans, it has been reported (Novikov and Rezanov 1962) that more than 99% of the ingested uranium remains in the body. Table III-7 shows the data for 2- to 3-year-old children for two different drinking water supplies. It is suspected that there must be some error in this Russian study and that, although children may have higher uptakes of uranium, the retention is less, leading to a dose rate that is relatively independent of age.

Table III-7 Uptake and Retention of Uranium

	Source A	Source B
Intake assuming an ingestion rate of 2 L/day (micrograms/day)	72	3.2
Fecal excretion (micrograms/day)	0.017	0.018
Urine concentration (micrograms/day)	0.033	0.0006
Percent of intake retained	99+%	99+%

From Novikov and Rezanov (1962).

In summary, the ICRP 30 model (1979) uses the following parameters to describe uranium metabolism in man:

f_1 = 0.05

f_2 for bone = 0.2

f_2 for kidney = 0.12

$t_{1/2}$ for kidney = 6 days

A more recent thorough review of the literature by Wrenn et al. (1985) suggests the following alternative values:

f_1 = 0.014
f_2 for bone = 0.2

f_2 for kidney = 0.11

$t_{1/2}$ for kidney = 15 days

IV. HUMAN EXPOSURE

A. Uranium Concentration in Drinking Water

Only a few drinking water sources have been directly analyzed for uranium. In a recent report discussed below (Drury et al. 1981), the drinking water concentrations are inferred from nearby ground and surface water. Some direct measurements have been made by the EPA and the Canadian(CRPB) Radiation Protection Bureau and will be discussed further.

The data collected by the EPA as required by the drinking water radiation regulations (Federal Register, Friday, July 9, 1976, p. 28402) are tabulated in Table IV-1. The values listed in Table IV-1 are not necessarily a complete list. Uranium measurements are not required by present regulations because the present MCL for gross alpha particle activity specifically excludes uranium and radon. However, since the drinking water radiation regulations allow uranium activity to be subtracted from the level of gross alpha particle activity for compliance purposes, some water supplies did analyze for uranium. As can be seen, many of these concentrations exceeded the level of 10 pCi/L.

The Office of Radiation Programs (ORP) of the USEPA has analyzed some selected drinking water samples for uranium. As can be seen from Table IV-2, the concentrations are usually

Table IV-1 Uranium Concentrations in Some U.S. Water Supplies[a]

Location	Uranium concentration (pCi/L)
Komvo, Arizona	18 ± 2, 15 ± 1
Choulic, Arizona	17 ± 2, 23 ± 2
Lower Santon, Arizona	44 ± 2, 49 ± 2
Cove Community, Arizona	52 ± 2, 55 ± 3, 57 ± 3
Red Mesa Public School, Arizona	20 ± 2, 8 ± 0.7, 15 ± 0.8
Sweetwater, Arizona	
Solar system	15 ± 4, 19 ± 2, 10 ± 1
Dry mesa	34 ± 4, 22 ± 2
Badger, California	8.5, 13.3
Lathrop, California	113, 44, 48
Camp Antelope, California	18 ± 1
Winter Haven Wells, Florida	0.8
Avon Park, Florida	
Franklin St. well	0.5
Areadia well I	0.6
Bowling Green, Florida	0.6
Plant City, Florida, Well 2	0.8
Zephyrillis, Florida	0.8
Clermont, Florida, Highland well	1.2
Mulberry, Florida, Well 1	0.9
Polk County, Florida	0.5, 1.0, 0.6, 0.07

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[a] This data was collected as a result of the existing regulations for radioactivity in drinking water.

Table IV-1 - (continued)

Location	Uranium concentration (pCi/L)
Polk County, Florida, Private wells	0.9, 7.3, 2.8, 1.6, 0.6, 0.9, 1.1
Hardee County, Florida	0.5
Orange County, Florida	0.06
Hillsborough County, Florida	0.9
Lake Arrowhead, Cherokee County, Georgia	2 ± 1
Seminole Mobile Home Park, Georgia (Clark County)	12 ± 1.2
Doublee Mobile Home Park, Georgia (Clayton County)	7 ± 1
Royal Court Mobile Home Park, VA (Clayton County)	26 ± 2
Cleaton Mobile Home Park, Georgia (Heard County)	2 ± 1
Meadow Haven Mobile Home Park, Georgia, (Heard County)	2 ± 1
Pine Haven Mobile Home Park, Georgia, (Heard County)	14 ± 3
Pomona Mobile Home Park, Georgia (Spalding County)	38 ± 6
County Haven Mobile Home Park, (Tift County)	2 ± 1
Priscses Mobile Home Park, Georgia (Walter County)	7 ± 2
Jonesboro, Georgia	92 ± 12

Table IV-1 - (continued)

Location	Uranium concentration (pCi/L)
Senoia, Georgia	27 ± 6
Attapulgus, Georgia	4 ± 2
Brooks, Georgia	50 ± 3
Brittany Harbor, Georgia	2 ± 1
Brittany Harbor North, Georgia	14 ± 2.8
Brittany Harbor West, Georgia	2 ± 1
White Plains, Georgia	8 ± 2
Grayson, Georgia	4 ± 2
Sugar Hill, Georgia	11 ± 1
Franklin, Georgia	10 ± 3
Nicholson, Georgia	67 ± 6.7
Gray, Georgia	2 ± 1
Buena Vista, Georgia	2 ± 1
Warm Springs, Georgia	2 ± 1
Ailey, Georgia	2 ± 1
Mt. Vernon, Georgia	2 ± 1
Lexington, Georgia	3 ± 1
Maveys, Georgia	18 ± 1
Molena, Georgia	29 ± 6
Jersey, Georgia	41 ± 6
Camak, Georgia	72 ± 14

Table IV-1 - (continued)

Location	Uranium concentration (pCi/L)
Napa, Idaho	
Sunny Ridge I	16 ± 2
Sunny Ridge II	16 ± 2
Lake Ridge Subdivision	13 ± 2
Elder Subdivision	9 ± 2
Cherry Lanes Acres	65 ± 3
Ridgeview	28 ± 3
Twin Bridges	8 ± 2
Caldwell, Idaho	
Rich Subdivision	21 ± 2
R&R Subdivision	59 ± 2
Rosewood Subdivision	17 ± 3
Kimpton Acres III	49 ± 2
Valley View Apts.	12 ± 2
Gerlach, Nevada	73 ± 3
Webb County, Texas, Bruni Waterworks	15.6

Adapted from USEPA (1976).

Table IV-2 Uranium Concentration in Selected Drinking Water Samples[a]

	July 1973 to June 1974 Composite		
	^{234}U	^{235}U	^{238}U
AL: Montgomery	0.026±0.017	-----------	0.019±0.13
CA: Berkeley	0.059±0.029	-----------	0.034±0.021
CA: Los Angeles	2.20±0.35	0.0171±0.029	1.97±0.33
CO: Denver	1.45±0.25	0.041±0.021	0.86±0.16
FL: Miami	-----------	-----------	-----------
ID: Idaho Falls	0.945±0.16	0.021±0.011	0.484±0.087
IL: Chicago	0.077±0.025	-----------	0.056±0.021
ND: Bismarck	-----------	-----------	-----------
NM: Santa Fe	-----------	-----------	-----------
NV: Las Vegas	3.20±0.51	0.103±0.080	2.11±0.36
NY: Buffalo	-----------	-----------	-----------
NY: New York City	-----------	-----------	-----------
OH: Cincinnati	-----------	-----------	-----------
OK: Oklahoma City	-----------	-----------	-----------
OR: Portland	-----------	-----------	-----------
PA: Harrisburg	-----------	-----------	-----------
PA: Pittsburgh	-----------	-----------	-----------
SC: Anderson	0.017±0.009	-----------	0.015±0.009
SC: Columbia	-----------	-----------	-----------
VA: Lynchburg	0.016±0.010	-----------	0.022±0.012
TN: Knoxville	0.054±0.021	-----------	0.041±0.018

[a] Measured in pCi/L

Table IV-2 - (continued)

	July 1974 to June 1975 Composite		
	^{234}U	^{235}U	^{238}U
AL: Montgomery	0.039±0.012	0.002±0.003	0.036±0.11
CA: Berkeley	0.013±0.006	0	0.008±0.004
CA: Los Angeles	1.99±0.24	0.086±0.018	1.72±0.21
CO: Denver	0.160±0.027	0.005±0.004	0.109±0.023
FL: Miami	-----------	-----------	-----------
ID: Idaho Falls	0.800±0.112	0.030±0.010	0.436±0.065
IL: Chicago	0.080±0.018	0.004±0.004	0.72±0.017
ND: Bismarck	0.253±0.038	0.011±0.006	0.149±0.027
NM: Santa Fe	2.49±0.320	0.060±0.015	0.853±0.119
NV: Las Vegas	2.49±0.320	0.138±0.028	1.57±0.220
NY: Buffalo	0.080±0.010	0.006±0.004	0.074±0.027
NY: New York City	0.010±0.005	0.001±0.002	0.005±0.004
OH: Cincinnati	0.006±0.004	0.001±0.002	0.007±0.004
OK: Oklahoma City	0.025±0.009	0.001±0.002	0.014±0.006
OR: Portland	0.009±0.005	0.001±0.002	0.004±0.003
PA: Harrisburg	0.006±0.004	0.003±0.003	0.005±0.003
PA: Pittsburgh	0.025±0.009	0.001±0.002	0.016±0.007
SC: Anderson	0.009±0.005	0.001±0.002	0.008±0.004
SC: Columbia	0.014±0.006	0.001±0.002	0.010±0.005
TN: Knoxville	0.078±0.016	0.007±0.004	0.059±0.013
VA: Lynchburg	0.010±0.005	0.001±0.002	0.011±0.006

Table IV-2 - (continued)

		June 1975 to June 1976 Composite		
		^{234}U	^{235}U	^{238}U
AL:	Montgomery	0.027±0.010	0	0.028±0.010
CA:	Berkeley	0.039±0.015	0.004±0.004	0.022±0.011
CA:	Los Angeles	2.100±0.21	0.071±0.016	1.87±0.21
CO:	Denver	1.25±0.16	0.038±0.011	1.00±0.14
FL:	Miami	0.120±0.022	0.007±0.005	0.104±0.020
ID:	Idaho Falls	0.828±0.099	0.014±0.006	0.433±0.056
IL:	Chicago	0.110±0.025	0.006±0.005	0.093±0.023
ND:	Bismarck	0.101±0.030	0.004±0.004	0.076±0.017
NM:	Santa Fe	-----------	-----------	-----------
NV:	Las Vegas	2.48±0.27	0.130±0.022	1.68±0.29
NY:	Buffalo	0.099±0.018	0.003±0.003	0.108±0.019
NY:	New York City	-----------	-----------	-----------
OH:	Cincinnati	0.134±0.062	0.021±0.021	0.077±0.044
OK:	Oklahoma City	0.039±0.016	0.003±0.003	0.019±0.011
OR:	Portland	0.045±0.014	0.002±0.002	0.009±0.005
PA:	Harrisburg	0.016±0.006	0.002±0.002	0.009±0.005
PA:	Pittsburgh	0.020±0.007	0.003±0.003	0.014±0.006
SC:	Anderson	0.030±0.009	-----------	0.029±009
SC:	Columbia	0.016±0.006	0.003±0.003	0.012±0.006
TN:	Knoxville	0.031±0.010	0.003±0.003	0.016±0.007
VA:	Lynchburg	0.018±0.002	0.001±0.002	0.006±0.004

Table IV-2 - (continued)

		July 1976 to June 1977 Composite		
		^{234}U	^{235}U	^{238}U
AL:	Montgomery	0.009±0.005	0000	0.008±0.001
CA:	Berkeley	0.031±0.10	0.003±0.003	0.019±0.008
CA:	Los Angeles	2.030±0.240	0.069±0.015	1.810±0.240
CO:	Denver	1.010±0.120	0.036±0.010	0.707±0.092
FL:	Miami	0.099±0.021	0.005±0.004	0.095±0.018
ID:	Idaho Falls	0.820±0.107	0.029±0.010	0.465±0.070
IL:	Chicago	-----------	-----------	-----------
ND:	Bismarck	0.129±0.022	0.009±0.005	0.090±0.017
NM:	Santa Fe	2.420±0.610	0.056±0.034	0.659±0.165
NV:	Las Vegas	2.500±0.300	0.080±0.016	1.570±0.190
NY:	Buffalo	0.153±0.028	0.005±0.006	0.113±0.023
NY:	New York City	-----------	-----------	-----------
OH:	Cincinnati	0.038±0.010	0.002±0.002	0.033±0.009
OK:	Oklahoma City	0.034±0.011	0.003±0.003	0.028±0.010
OR:	Portland	0.013±0.006	0.001±0.002	0.007±0.004
PA:	Harrisburg	0.038±0.019	0.000	0.040±0.020
PA:	Pittsburgh	0.044±0.011	0.004±0.003	0.025±0.008
SC:	Anderson	-----------	-----------	-----------
SC:	Columbia	0.017±0.007	0.003±0.003	0.018±0.007
TN:	Knoxville	-----------	-----------	-----------
VA:	Lynchburg	0.021±0.008	0.003±0.003	0.017±0.007

Table IV-2 - (continued)

		July to December 1977 Composite		
		^{234}U	^{235}U	^{238}U
AL:	Montgomery	0.028±0.009	0.002±0.003	0.014±0.006
CA:	Berkeley	0.018±0.007	0.007±0.005	0.018±0.007
CA:	Los Angeles	1.470±0.190	0.063±0.017	1.400±0.180
CO:	Denver	1.210±0.160	0.096±0.019	0.974±0.130
FL:	Miami	0.091±0.026	0.007±0.007	0.083±0.025
ID:	Idaho Falls	-----------	-----------	-----------
IL:	Chicago	0.182±0.064	0.015±0.015	0.182±0.044
ND:	Bismarck	0.065±0.018	0.004±0.004	0.054±0.015
NM:	Santa Fe	0.221±0.042	0.002±0.003	0.080±0.018
NV:	Las Vegas	3.150±0.380	0.111±0.024	2.080±0.270
NY:	Buffalo	0.142±0.024	0.006±0.004	0.115±0.021
NY:	New York City	-----------	-----------	-----------
OH:	Cincinnati	0.035±0.011	0.002±0.003	0.028±0.009
OK:	Oklahoma City	0.092±0.020	0.003±0.003	0.075±0.020
OR:	Portland	0.015±0.006	0.002±0.003	0.011±0.005
PA:	Harrisburg	0.016±0.007	0.003±0.003	0.011±0.006
PA:	Pittsburgh	0.081±0.017	0.004±0.004	0.056±0.013
SC:	Anderson	0.025±0.008	0.002±0.003	0.020±0.007
SC:	Columbia	0.017±0.007	0.001±0.002	0.009±0.005
TN:	Knoxville	0.036±0.011	0.001±0.002	0.025±0.009
VA:	Lynchburg	0.031±0.012	0.003±0.003	0.031±0.031

Table IV-2 - (continued)

		July 1976 to June 1977 Composite		
		^{234}U	^{235}U	^{238}U
AL:	Montgomery	0.009±0.005	0000	0.008±0.001
CA:	Berkeley	0.031±0.10	0.003±0.003	0.019±0.008
CA:	Los Angeles	2.030±0.240	0.069±0.015	1.810±0.240
CO:	Denver	1.010±0.120	0.036±0.010	0.707±0.092
FL:	Miami	0.099±0.021	0.005±0.004	0.095±0.018
ID:	Idaho Falls	0.820±0.107	0.029±0.010	0.465±0.070
IL:	Chicago	-----------	-----------	-----------
ND:	Bismarck	0.129±0.022	0.009±0.005	0.090±0.017
NM:	Santa Fe	2.420±0.610	0.056±0.034	0.659±0.165
NV:	Las Vegas	2.500±0.300	0.080±0.016	1.570±0.190
NY:	Buffalo	0.153±0.028	0.005±0.006	0.113±0.023
NY:	New York City	-----------	-----------	-----------
OH:	Cincinnati	0.038±0.010	0.002±0.002	0.033±0.009
OK:	Oklahoma City	0.034±0.011	0.003±0.003	0.028±0.010
OR:	Portland	0.013±0.006	0.001±0.002	0.007±0.004
PA:	Harrisburg	0.038±0.019	0.000	0.040±0.020
PA:	Pittsburgh	0.044±0.011	0.004±0.003	0.025±0.008
SC:	Anderson	-----------	-----------	-----------
SC:	Columbia	0.017±0.007	0.003±0.003	0.018±0.007
TN:	Knoxville	-----------	-----------	-----------
VA:	Lynchburg	0.021±0.008	0.003±0.003	0.017±0.007

Table IV-2 - (continued)

		July to December 1977 Composite		
		^{234}U	^{235}U	^{238}U
AL:	Montgomery	0.028±0.009	0.002±0.003	0.014±0.006
CA:	Berkeley	0.018±0.007	0.007±0.005	0.018±0.007
CA:	Los Angeles	1.470±0.190	0.063±0.017	1.400±0.180
CO:	Denver	1.210±0.160	0.096±0.019	0.974±0.130
FL:	Miami	0.091±0.026	0.007±0.007	0.083±0.025
ID:	Idaho Falls	-----------	-----------	-----------
IL:	Chicago	0.182±0.064	0.015±0.015	0.182±0.044
ND:	Bismarck	0.065±0.018	0.004±0.004	0.054±0.015
NM:	Santa Fe	0.221±0.042	0.002±0.003	0.080±0.018
NV:	Las Vegas	3.150±0.380	0.111±0.024	2.080±0.270
NY:	Buffalo	0.142±0.024	0.006±0.004	0.115±0.021
NY:	New York City	-----------	-----------	-----------
OH:	Cincinnati	0.035±0.011	0.002±0.003	0.028±0.009
OK:	Oklahoma City	0.092±0.020	0.003±0.003	0.075±0.020
OR:	Portland	0.015±0.006	0.002±0.003	0.011±0.005
PA:	Harrisburg	0.016±0.007	0.003±0.003	0.011±0.006
PA:	Pittsburgh	0.081±0.017	0.004±0.004	0.056±0.013
SC:	Anderson	0.025±0.008	0.002±0.003	0.020±0.007
SC:	Columbia	0.017±0.007	0.001±0.002	0.009±0.005
TN:	Knoxville	0.036±0.011	0.001±0.002	0.025±0.009
VA:	Lynchburg	0.031±0.012	0.003±0.003	0.031±0.031

Adapted from USEPA (1980)

less than 1 pCi/L, and the highest measured concentration was 3.2 pCi/L (USEPA 1980). Monitoring data from EPA analysis of Navajo Indian Reservation drinking water supplies are shown in Table IV-3.

In 68 stations in Canada, the range in concentrations in surface water was from 0.0001 mg/L (0.067 pCi/L) to 0.0043 mg/L (2.9 pCi/L). In Canada, no monthly composites for the drinking water in several cities exceeded 0.006 mg/L (4 pCi/L), and they were usually less than 0.001 mg/L (0.67 pCi/L) (CRPB 1980). Some typical values are shown in Table IV-4. Table IV-5 shows some uranium concentrations in other natural water around the world.

A recent study by Oak Ridge National Laboratory (Drury et al. 1981) reported the analyses of 89,944 U.S. surface, ground and domestic waters. Those ground and surface water sources that are used for drinking water were called domestic water in this study. Most of these samples were taken as a result of the National Uranium Resource Evaluation (NURE) program. The samples were taken in a grid of the U.S. set up to block off areas of the order of 10 to 15 km^2. The samples were from surface and groundwaters, and only a fraction of all the possible samples have been collected and analyzed. In addition, data were found for uranium in most large city water supplies. A summary of the Oak Ridge report is shown in Tables IV-6, IV-7 and IV-8.

Table IV-3 Monitoring Data from Drinking Water on the Navajo Reservation

Location	U Concentration (pCi/L)
Sweetwater, AZ Chapter House	12.2
Sweetwater, AZ Solar	18.5
Many Farms, AZ BIA #3	4.1
Many Farms, AZ Elementary School	3.1
Round Rock, AZ Chapter House	4.3
Cove, AZ (12T-341)	38.7
Red (Rock) Valley, AZ Chapter House	12.8
Smith Lake, NM Chapter House	9.4
Smith Lake, NM (16T-593)	13.4
Martinez Camp, NM	6.0
Marino Lake, NM Chapter House	13.7
Church Rock, NM Elementary School	2.7

Adapted from USEPA (1980c).

Table IV-4 Uranium in Canadian Drinking Water (micrograms/L)

	From June 1975 to December 1976	
Municipality	No.[a]	Maximum[b]
Victoria, B.C.	6	0.57
Vancouver, B.C.	4	0.28
Edmonton, Alta.	11	0.26
Calgary, Alta.	14	0.52
Saskatoon, Sask.	18	4.30
Regina, Sask.	19	4.40
Winnipeg, Man.	5	0.24
Sault St. Marie, Ont.	10	0.75
Sudbury, Ont.	4	0.38
Toronto, Ont.	11	0.38
Ottawa, Ont.	9	0.33
Montreal, Que.	7	0.42
Quebec, Que.	6	0.56
Saint John, N.B.	7	0.28
Halifax, N.S.	5	0.19
St. John's, Nlfd.	8	0.42
Elliot Lake, Ont.	17	2.40

[a] Number of monthly samples with concentrations above the detection limit of 0.05 micrograms/L.

[b] Highest concentration observed in a monthly sample in micrograms/L.

Adapted from CRPB (1980).

Table IV-5 Uranium Concentrations in Natural Waters of the World

Aqueous system	Uranium concentration (micrograms/L)	Estimated mean concentration (micrograms/L)	References
Amazon	0.04 0.02	0.03	a b
LaPlata Parna	--	0.1	c
Congo	--	0.12	b
Yangtze	--	0.5	c
Ganges-Bramaputra	1.6 (mouth), 1.9, 4.1, 6.6	1.6	d
Missouri	1.83, 2.39 1.0, 0.10 0.31, 0.97	0.95+0.90	e
Mekong		0.1	c
Nile	--	0.1	c
St. Lawrence	--	0.02	e

[a] Moore (1967).
[b] Bertine et al. (1970).
[c] Sackett et al. (1973).
[d] Bhat and Krishawamy (1969).
[e] Rona and Urry (1952).
[f] Sackett and Cook (1969).
[g] Lopatkina (1964).
[h] Miyake et al. (1964).
[i] Fix (1956).

Table IV-5 - (continued)

Aqueous system	Uranium concentration (micrograms/L)	Estimated mean concentration (micrograms/L)	References
Other Rivers			
2 European	0.16, 0.64	0.40	b
6 Indian	0.01, 0.01, 0.58, 0.68, 1.08, 3.50	1.00-1.30	d
27 North American	-	0.78-0.75	
1 South American	0.23	-	b
100 Russian	0.07-7.0		g
10 Japanese	0.34-1.2	0.57	h
U.S. (300 surface & Groundwaters, W. & S.E. U.S.A.)	-	0.10	i

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[a] Moore (1967).
[b] Bertine et al. (1970).
[c] Sackett et al. (1973).
[d] Bhat and Krishawamy (1969).
[e] Rona and Urry (1952).
[f] Sackett and Cook (1969).
[g] Lopatkina (1964).
[h] Miyake et al. (1964).
[i] Fix (1956).

Adapted from Drury et al. (1981).

Table IV-6 Summary of Occurrence of Uranium in U.S. Ground, Surface and Domestic Waters.

	Surface water (pCi/L)	Ground water (pCi/L)	Domestic water[a] (pCi/L)
Range	0.01-582	0.01-653	0.07-653
Average	1.06	3.18	1.73
Median concentration range	0.1-0.2	0.2-0.5	0.1-0.2
Modal concentration range	0.2-0.5	2-5	0.1-0.2
Total no.	34,561	55,433	28,239

[a] The surface and groundwater supplies identified as domestic water sources.

Adapted from Drury et al. (1981).

Table IV-7 Concentration for Specific Cities in States with the Largest Average Uranium Concentrations

	Concentration (pCi/L)	Population served (1,000's)
South Dakota		
Aberdeen	7.3	26
Brookings	5.4	13.5
Rapid City	0.7	45
Sioux Falls	5.4	72.4
Watertown	7.3	15
Yorktown	5.4	12.5
Non-urban	7.3	481.8
Nevada		
Las Vegas Valley Water District	5.2	200
North Las Vegas	5.2	50.3
Reno-Sierra Pacific Power	5.3	109
Non-urban	1.5	129.4
New Mexico		
Albuquerque	2.6	250
Clovis-Southwest Public Service	3.4	28.5
Las Vegas	1.5	14.5
Los Alamos	0.007	32
Roswell	0.7	40
Santa Fe	0.6	41.7
Non-urban	3.4	610.4
California	2.3-3.4	19,971
Wyoming		
Non-urban	3.4	241

Adapted from Drury et al. (1981).

Table IV-8 Ranking of States with Largest Average Uranium Concentrations

Rank	Uranium concentration (pCi/L)	State
1	6.7	South Dakota
2	4.3	Nevada
3	2.9	New Mexico
4	2.7	California Wyoming
5	2.6	Texas
6	2.5	Arizona Oklahoma
7	1.8	Colorado Nebraska
8	1.4	Montana
9	1.2	Kansas
10	0.8	Idaho
11	0.7	West Virginia
12	0.6	Florida Indiana Washington
13	0.5	Iowa Utah
14	0.3	Alaska Delaware Illinois Missour New York North Dakota

Table IV-8 - (continued)

Rank	Uranium concentration (pCi/L)	State
15	0.2	Connecticut Main Michigan Minnesota South Carolina Wisconsin
16	0.1	Kentucky New Jersey Rhode Island
17	0.1	Alabama Arkansas District of Columbia Georgia Hawaii Louisiana Maryland Massachusetts Mississippi New Hampshire North Carolina Ohio Oregon Pennsylvania Tennessee Vermont Virginia

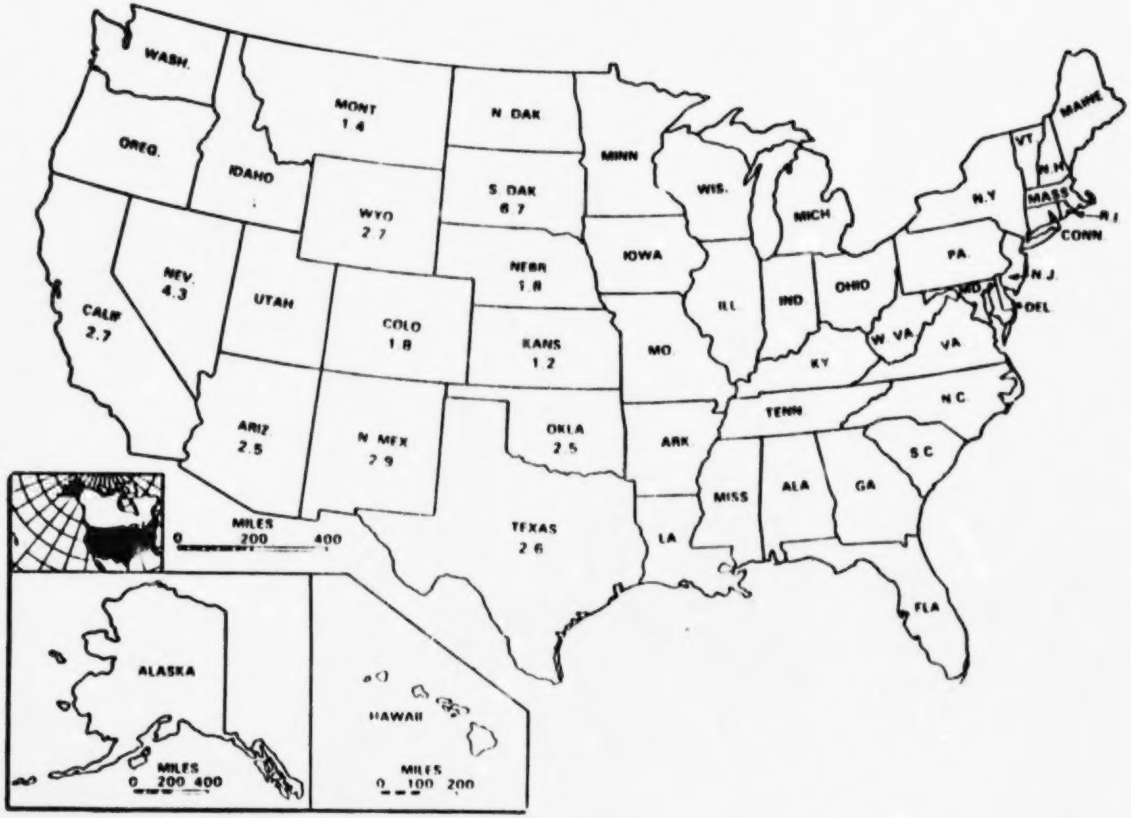
Adapted from Drury et al. (1981).

Figure IV-1 shows those states that exceed a population-weighted average of 1 pCi/L. The population-weighted average is the sum of the products of population and corresponding concentration, divided by the total population. Concentration histograms are shown in Figures IV-2 to IV-4.

In order to estimate the impact of a possible standard for uranium, two approaches were used. One assumed that the distribution of values for drinking water sources actually measured (usually only the larger suppliers) was representative of all the drinking water sources. An alternative approach assumed that the distribution of values for surface and groundwaters was representative. These two approaches produced a range of values: of the 63,975 public water supplies in the United States, 25 to 650 supplies exceeded a uranium concentration of 20 pCi/L, 100 to 2,000 supplies exceeded 10 pCi/L, and 2,500 to 5,000 supplies exceeded 5 pCi/L. The lower estimates are due to the first approach.

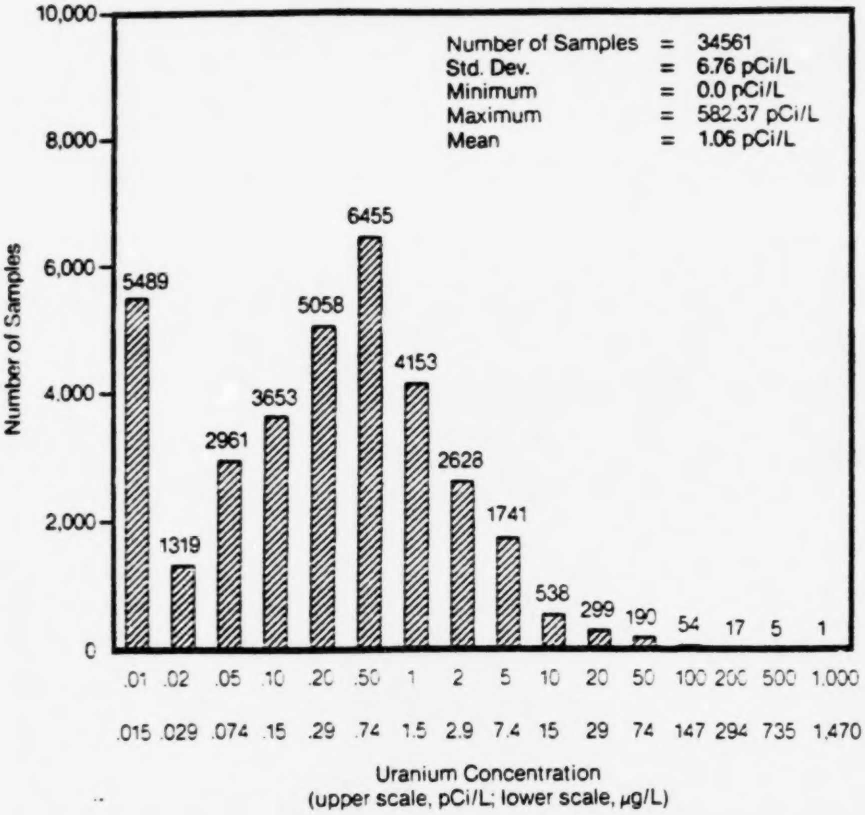
As can be seen in Table IV-7, most of the water supplies that exceeded 5 pCi/L are small towns on water supplies serving less than a few thousand persons.

Figure IV-5 shows where the samples that provided the information contained in the ORNL report (Drury et al. 1981) were taken. The dots represent individual locations, and the


WASH.
OREG.
CALIF
2.7
IDAHO
NEV.
4.3
MONT
1.4
WYO
2.7
UTAH
ARIZ
2.5
COLO
1.8
N MEX
2.9
N DAK
S DAK
6.7
NEBR
1.8
KANS
1.2
OKLA
2.5
TEXAS
2.6
MINN
IOWA
MO
ARK
LA
WIS.
ILL
MICH
IND
OHIO
KY
TENN
MISS
ALA
GA
FLA
S C
N C
VA
W VA
MD
DEL
N.J.
PA.
N.Y
CONN
R.I
MASS
N.H
VT
MAINE
MILES
0 200 400
ALASKA
MILES
0 200 400
HAWAII
MILES
0 100 200

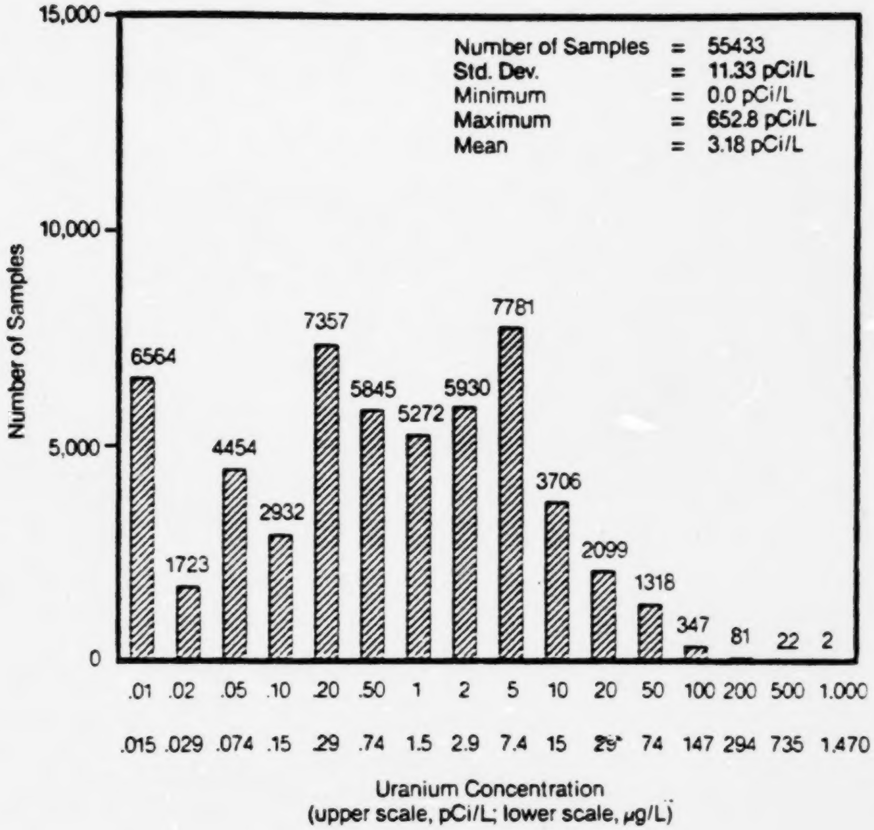
Adapted from Drury et al. (1981).

Figure IV-1 Average Uranium Water Concentrations for States Whose Concentration Exceeds 1 pCi/L



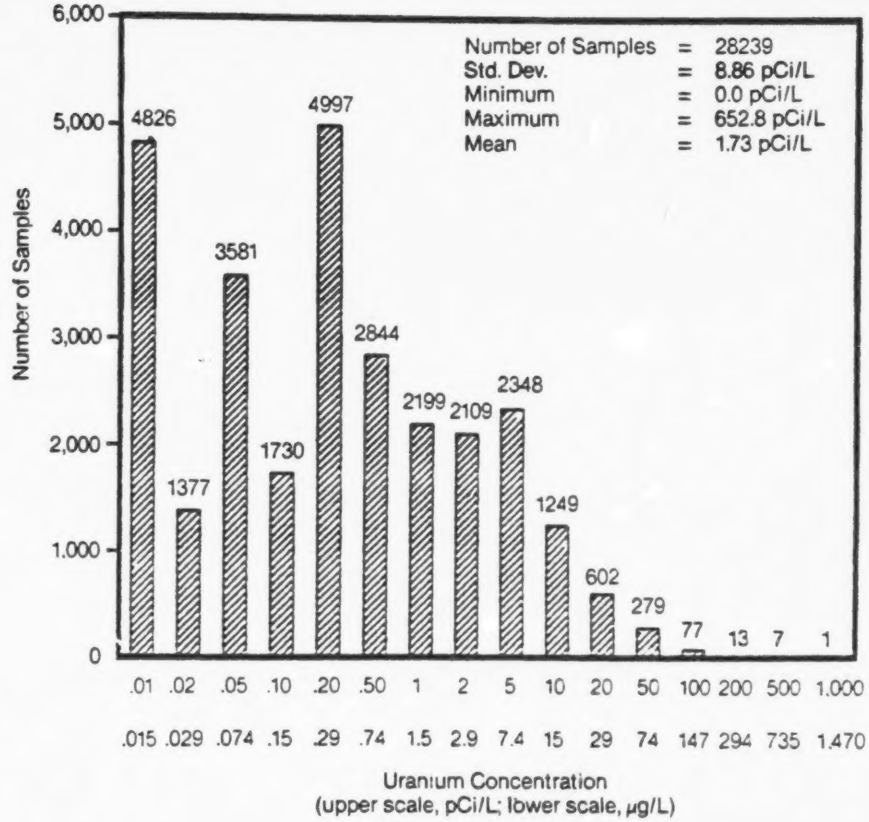
Adapted from Drury et al. (1981).

Figure IV-2 Histogram of Uranium Concentrations in Surface Waters of the United States



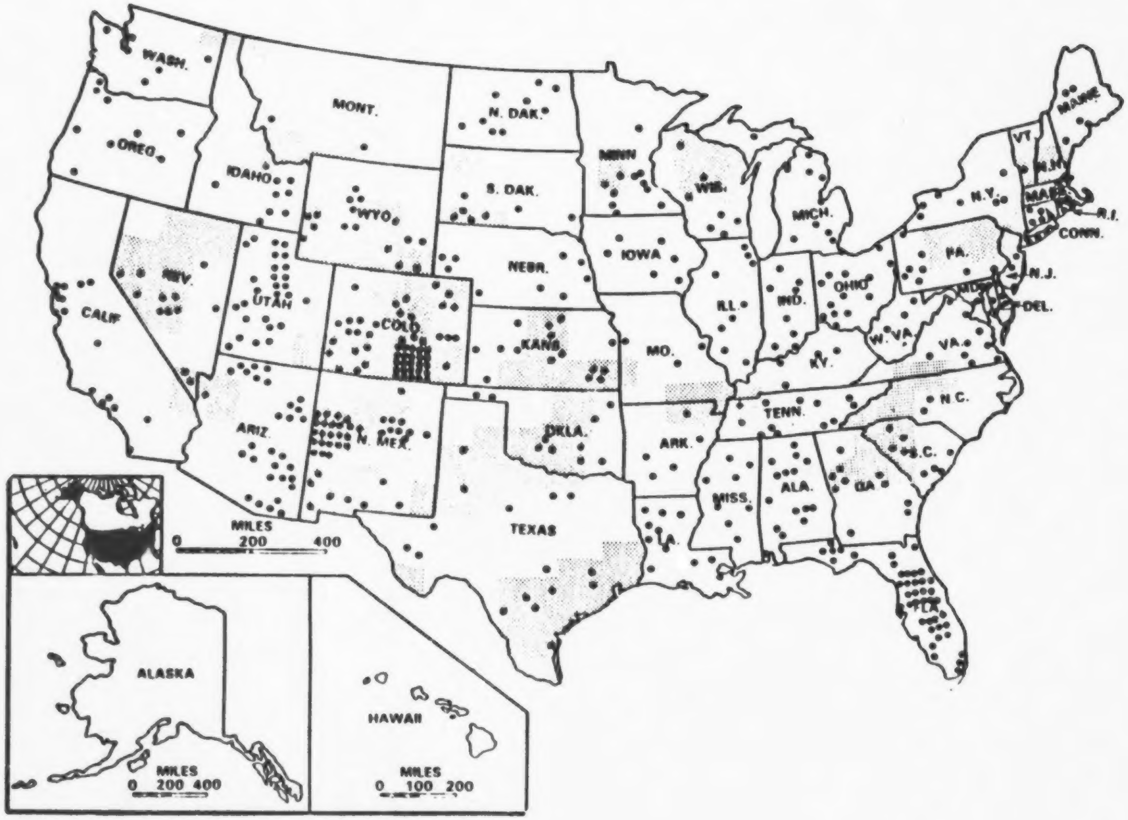
Adapted from Drury et al. (1981).

Figure IV-3 Histogram of Uranium Concentrations in Ground Waters of the United States



Adapted from Drury et al. (1981).

Figure IV-4 Histogram of Uranium Concentrations in Domestic Waters of the United States



WASH.
OREG.
CALIF.
IDAHO
NEV.
UTAH
ARIZ.
MONT.
WYO.
COLO.
N. MEX.
N. DAK.
S. DAK.
NEBR.
KANS.
OKLA.
TEXAS
MINN.
IOWA
MO.
ARK.
LA.
WIS.
ILL.
MISS.
MICH.
IND.
KY.
TENN.
ALA.
OHIO
W. VA.
GA.
FLA.
S.C.
N.C.
VA.
PA.
N.Y.
VT.
N.H.
MAINE
MASS.
R.I.
CONN.
N.J.
DEL.
MD.
MILES
0 200 400
ALASKA
MILES
0 200 400
HAWAII
MILES
0 100 200

Adapted from Drury et al. (1981).

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[a] The data are specific locations and the cross-hatched areas were sampled from 10 to 15 km^2 areas.

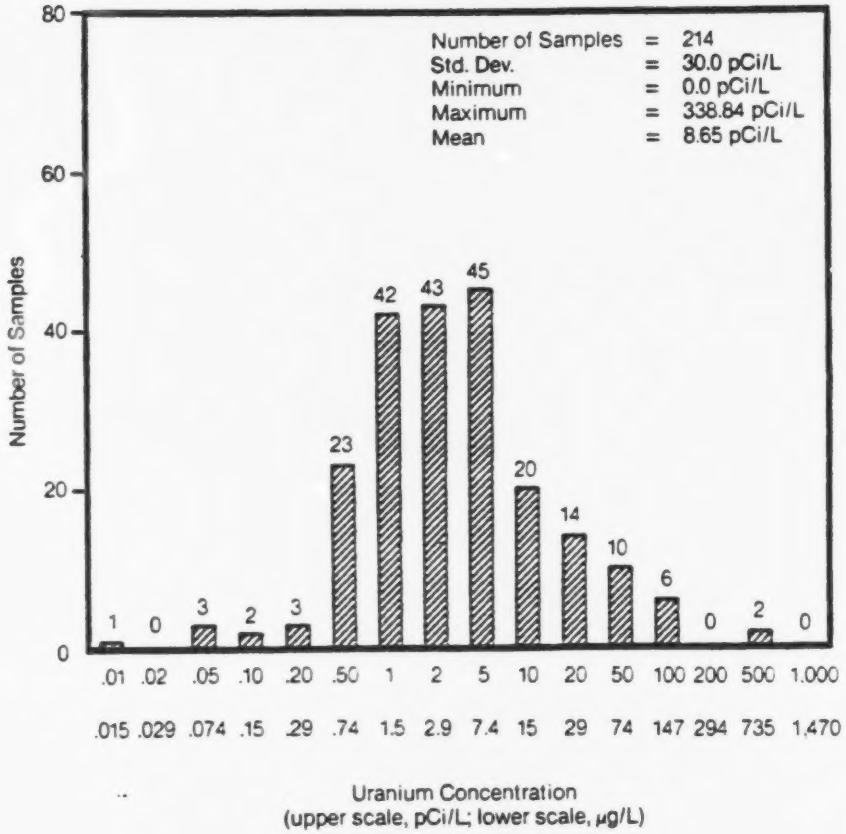
Figure IV-5 Locations for Which Data Regarding Uranium Water Concentrations Were Obtained[a]

cross-hatched area represents the NURE samples that have been analyzed. Figures IV-6 to IV-15 show histograms for the states with the larger concentrations of uranium in domestic water.

The high uranium concentration in waters in South Dakota would suggest that there is a large uranium deposit there. This possibility is consistent with the local geology, which is similar to that around Elliot Lake in Canada, where there is a known large deposit of uranium in the water (Cheney 1981).

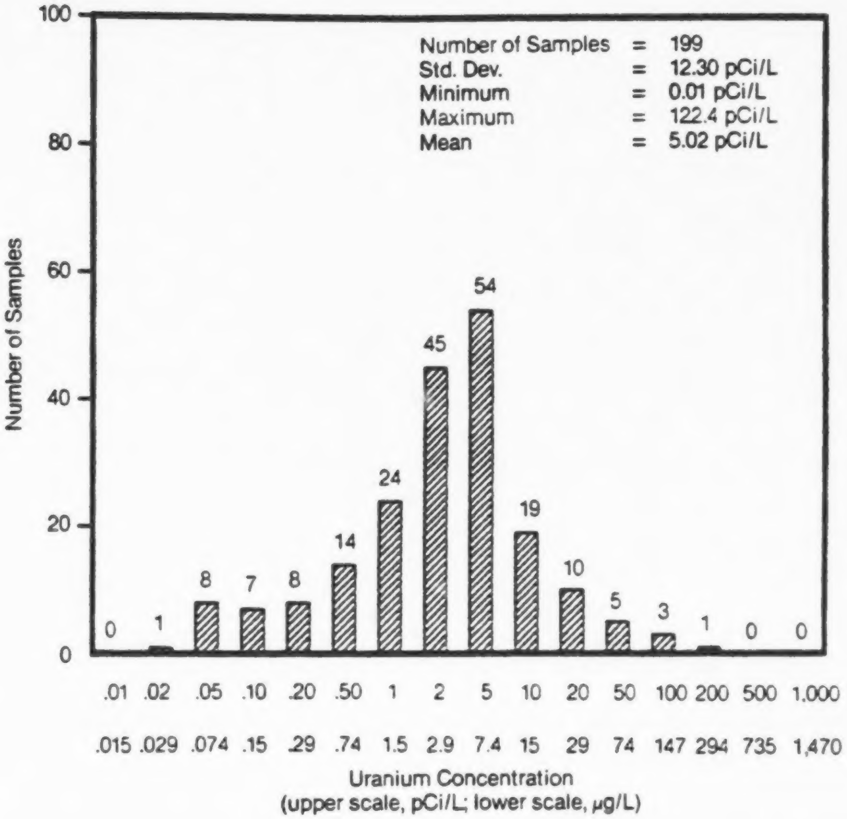
The ratio of ^{234}U to ^{238}U will be 1 for equilibrium. However, the ratio often exceeds 1. In the decay process, electrons can be preferentially stripped in the decay to ^{234}U allowing it to form the more soluble +6 valence state. It has been observed that the largest disequilibrium ratios occur in slightly oxidizing environments (Kaufman et al. 1969). The mobility of the intervening short-lived daughter ^{234}U may explain the range of ratios. In reducing environments, ^{234}U does not appear to be preferentially leached. The $^{234}U/^{238}U$ ratio is also a function of the time since the isotopes were leached, in that it is closer to 1 as time goes on (Turekian 1971).

It is possible for the $^{234}U/^{238}U$ ratio to be less than 1 if the source of uranium is weathered rock or materials that have been exposed to extensive groundwater circulation and leaching (Osmond et al. 1969).



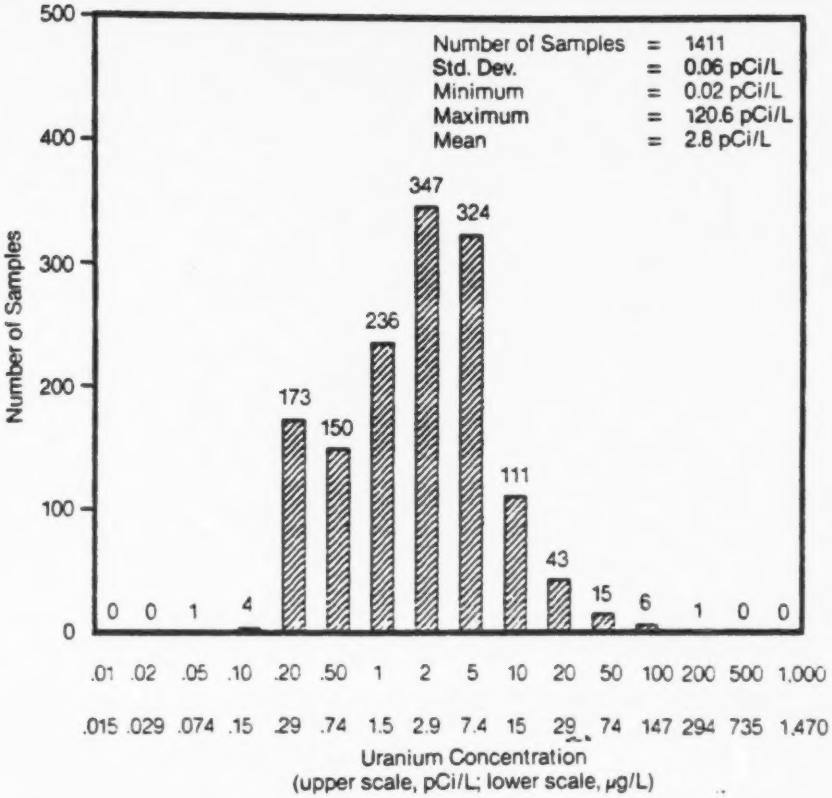
Adapted from Drury et al. (1981).

Figure IV-6 Histogram of Uranium Concentrations in Domestic Waters of Arizona



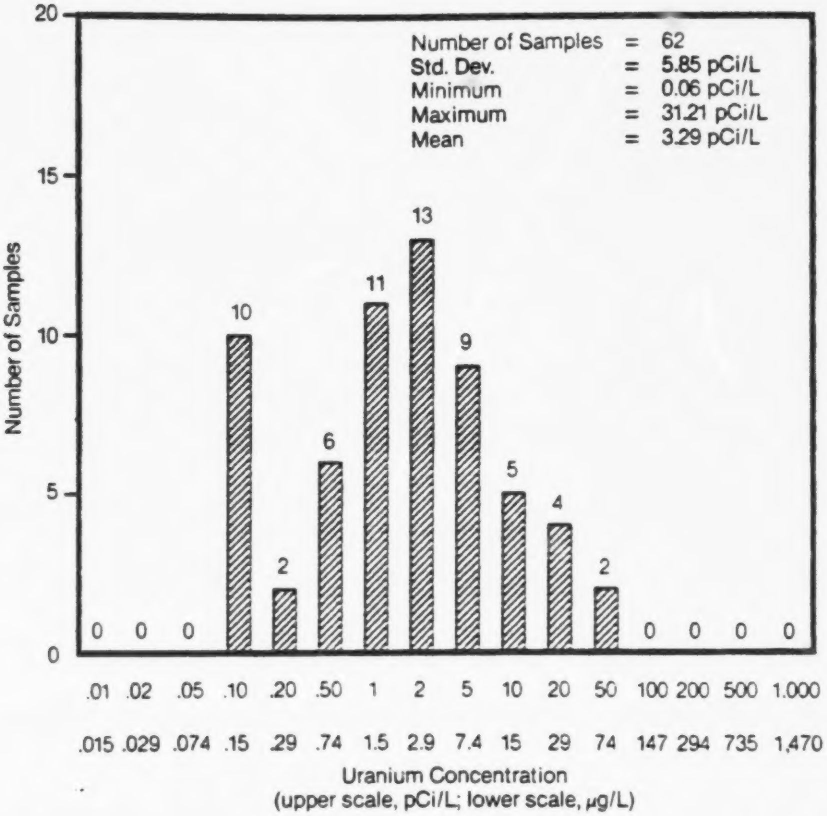
Adapted from Drury et al. (1981).

Figure IV-7 Histogram of Uranium Concentrations in Domestic Waters of Colorado



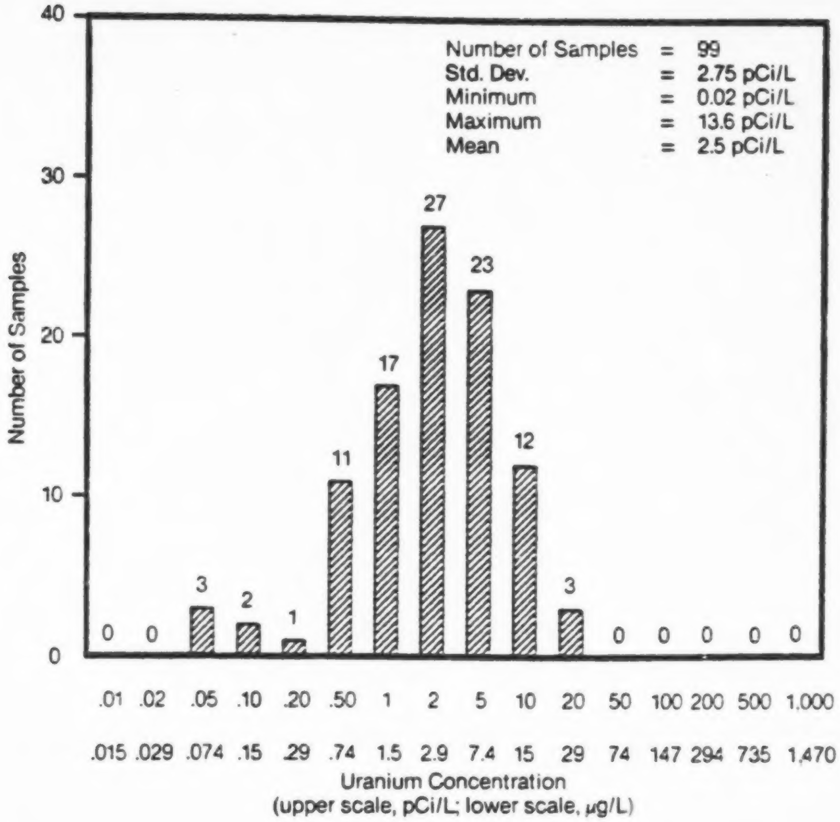
Adapted from Drury et al. (1981).

Figure IV-8 Histogram of Uranium Concentrations in Domestic Waters of Kansas



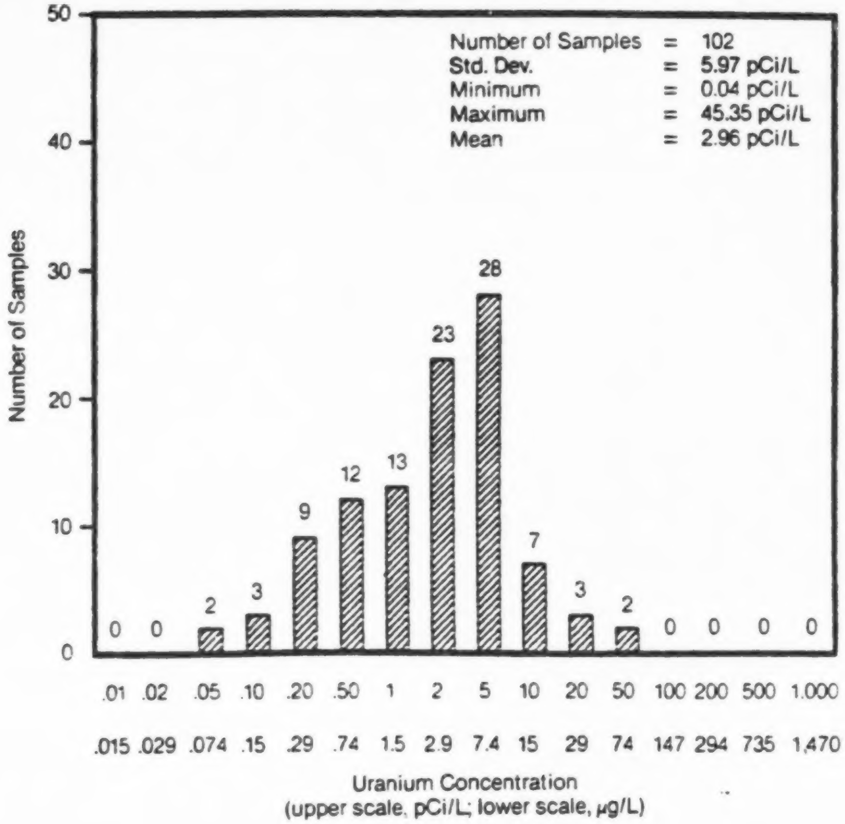
Adapted from Drury et al. (1981).

Figure IV-9 Histogram of Uranium Concentrations in Domestic Waters of Montana



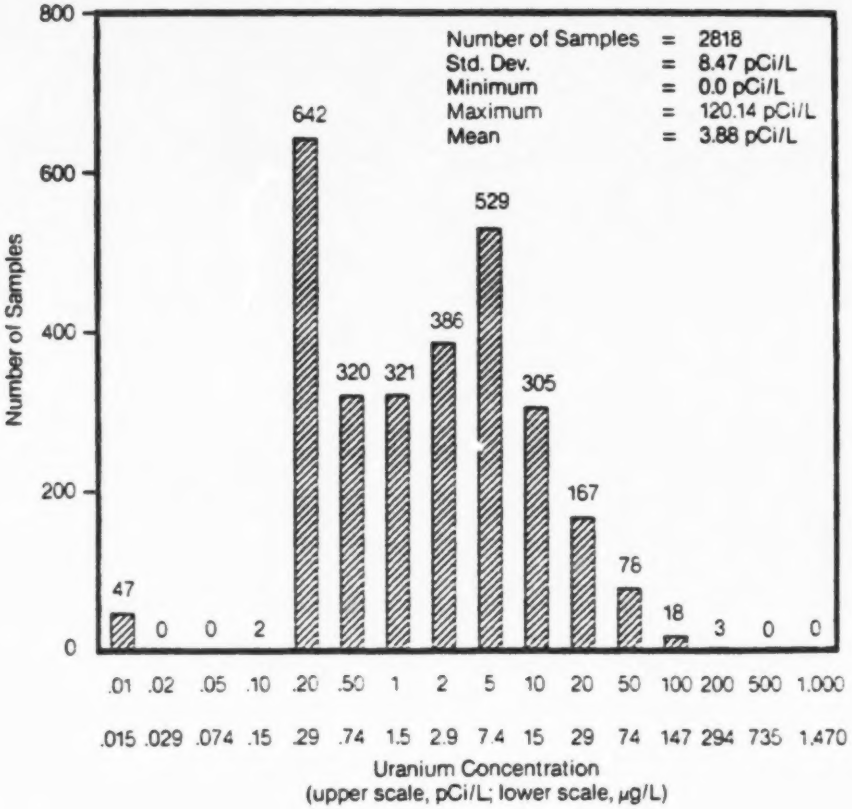
Adapted from Drury et al. (1981).

Figure IV-10 Histogram of Uranium Concentrations in Domestic Waters of Nevada



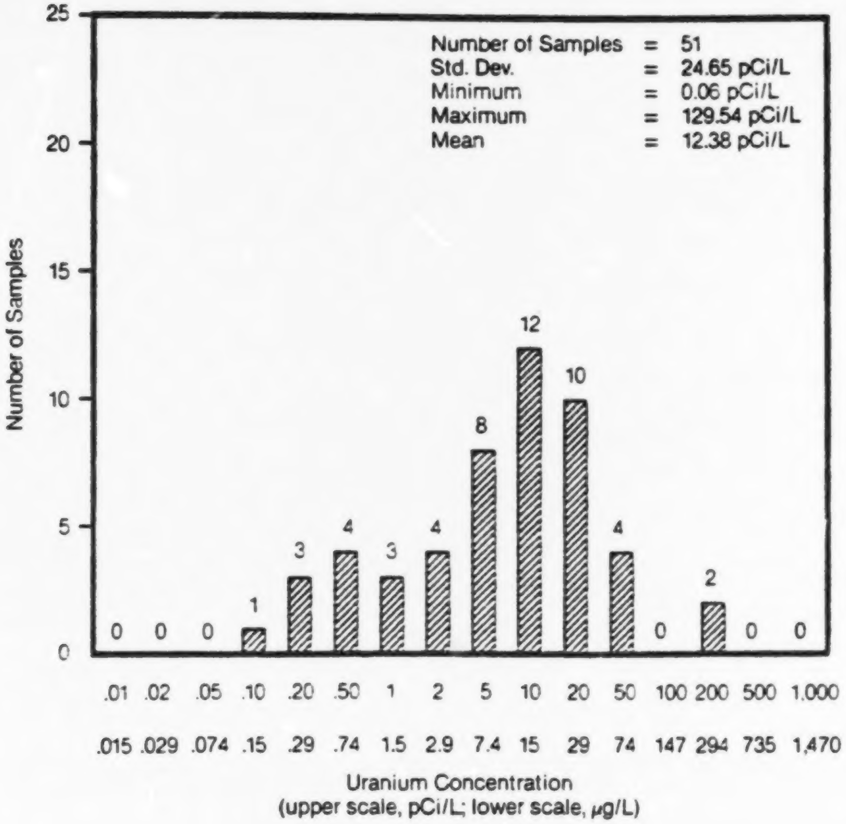
Adapted from Drury et al. (1981).

Figure IV-11 Histogram of Uranium Concentrations in Domestic Waters of New Mexico



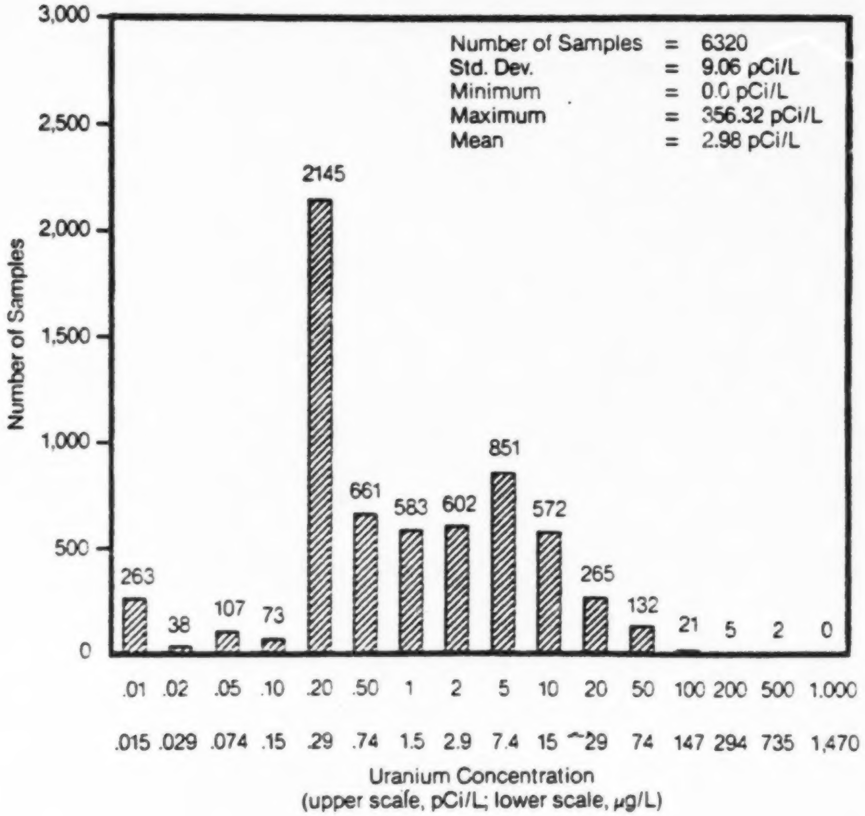
Adapted from Drury et al. (1981).

Figure IV-12 Histogram of Uranium Concentrations in Domestic Waters of Oklahoma



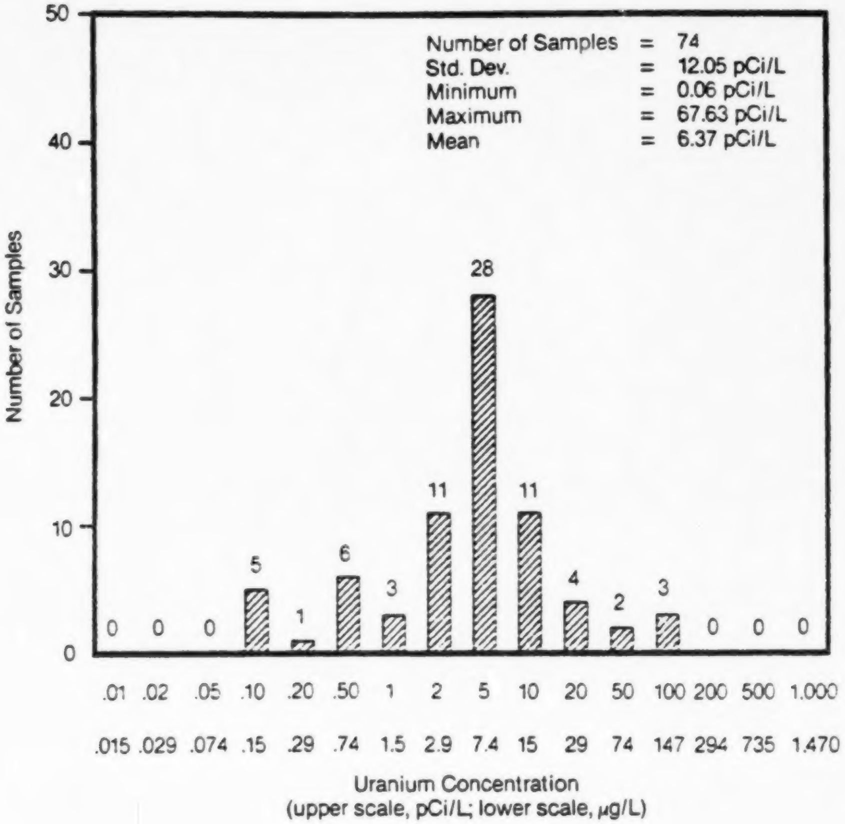
Adapted from Drury et al. (1981).

Figure IV-13 Histogram of Uranium Concentrations in Domestic Waters of South Dakota



Adapted from Drury et al. (1981).

Figure IV-14 Histogram of Uranium Concentrations in Domestic Waters of Texas



Adapted from Drury et al. (1981).

Figure IV-15 Histogram of Uranium Concentrations in Domestic Waters of Wyoming

As can be seen in Table IV-9 (USEPA 1980), the $^{234}U/^{238}U$ ratio rarely exceeds 2. A study of the Florida aquifer (Kaufman et al. 1969 and Osmond et al. 1974) showed ratios in the range 0.05 to 3.9. (This study involved a few hundred samples, with only one showing a ratio greater than 3.0.) Surface waters in central Texas had ratios in the range of 1.14 to 2.42. However, samples from the Cretaceous Trinity aquifer in central Texas showed values in the range 0.92 to 12.25 (Kronfeld 1974). The ratio for seawater seems established at 1.14.

The data for $^{234}U/^{238}U$ ratios are far from being complete or descriptive of ground and surface waters in the U.S. However, from the few data available, it appears that surface waters are closer to equilibrium for uranium isotopes than are groundwaters; only in a few cases does the ratio exceed 3, and only rarely does it exceed 9.

B. Uranium in the Environment

Uranium is known to be found in phosphate deposits and can come from tailings and phosphate deposits, as well as from runoff from phosphate fertilizers (Spalding and Sackett 1972). The average uranium concentration is about 100 micrograms/g (or 67 pCi/g). Measurements of phosphate materials from Florida indicate uranium concentrations in the following ranges: central Florida, 20.2 to 83.4 pCi/g; north Florida, 4.5 to 11.4

Table IV-9 $^{234}U/^{238}U$ Ratios

		July 1973-June 1974	July 1974-June 1975	July 1975-June 1976
AL:	Montgomery	1.4 ± 1.2	1.1 ± 0.4	1.0 ± 0.5
CA:	Berkeley	2.0 ± 1.5	1.6 ± 0.7	1.8 ± 1.1
CA:	Los Angeles	1.1 ± 0.2	1.2 ± 0.2	1.1 ± 0.2
CO:	Denver	1.7 ± 0.4	1.5 ± 0.3	1.3 ± 0.3
FL:	Miami	---------	---------	1.1 ± 0.3
ID:	Idaho Falls	1.9 ± 0.4	1.8 ± 0.2	1.9 ± 0.2
IL:	Chicago	1.4 ± 0.7	1.1 ± 0.3	1.2 ± 0.4
ND:	Bismarck	---------	1.7 ± 0.4	1.3 ± 0.5
NM:	Santa Fe	---------	2.9 ± 0.6	---------
NV:	Las Vegas	1.5 ± 0.3	1.6 ± 0.3	1.5 ± 0.3
NY:	Buffalo	---------	1.1 ± 0.4	0.9 ± 0.3
NY:	New York City	---------	2.0 ± 1.7	---------
OH:	Cincinnati	---------	1.8 ± 1.6	1.7 ± 1.2
OK:	Oklahoma City	---------	2.2 ± 1.1	2.1 ± 1.5
OR:	Portland	---------	2.2 ± 1.8	2.1 ± 1.5
PA:	Harrisburg	---------	1.2 ± 1.0	1.8 ± 1.2
PA:	Pittsburgh	---------	1.6 ± 0.9	1.4 ± 0.8
SC:	Anderson	1.1 ± 0.5	1.1 ± 1.0	1.0 ± 0.4
SC:	Columbia	---------	1.4 ± 0.9	1.3 ± 0.8
TN:	Knoxville	1.3 ± 0.8	1.3 ± 0.4	1.9 ± 1.0
VA:	Lynchburg	0.7 ± 0.7	0.9 ± 0.8	3.0 ± 2.0

Table IV-9 - (continued)

	July 1976-June 1977	July - December 1977
AL: Montgomery	1.1 ± 0.7	2.0 ± 1.1
CA: Berkeley	1.6 ± 0.9	1.0 ± 0.5
CA: Los Angeles	1.1 ± 0.2	1.1 ± 0.2
CO: Denver	1.4 ± 0.2	1.2 ± 0.2
FL: Miami	1.0 ± 0.3	1.1 ± 0.4
ID: Idaho Falls	1.8 ± 0.4	---------
IL: Chicago	---------	1.0 ± 0.4
ND: Bismarck	1.4 ± 0.4	1.2 ± 0.4
NM: Santa Fe	3.7 ± 1.3	2.8 ± 0.8
NV: Las Vegas	1.6 ± 0.3	1.5 ± 0.3
NY: Buffalo	1.4 ± 0.4	1.2 ± 0.3
NY: New York City	---------	---------
OH: Cincinnati	1.1 ± 0.4	1.3 ± 0.4
OK: Oklahoma City	1.2 ± 0.6	1.2 ± 0.4
OR: Portland	1.9 ± 1.4	1.4 ± 0.8
PA: Harrisburg	1.0 ± 0.7	1.4 ± 0.8
PA: Pittsburgh	1.8 ± 0.7	1.4 ± 0.4
SC: Anderson	---------	1.3 ± 0.6
SC: Columbia	1.0 ± 0.6	1.9 ± 1.3
TN: Knoxville	---------	1.4 ± 0.7
VA: Lynchburg	1.2 ± 0.7	1.0 ± 1.0

Adapted from USEPA (1980).

pCi/g (Rona 1956). When processed, the uranium tends to follow the phosphoric acid (the radium found in phosphate deposits tends to follow the by-product gypsum). The uranium content of the resulting fertilizers is 25.3 pCi/g for the ammoniate phosphate and 26.0 Ci/g for the triple superphosphate.

The use of phosphate fertilizers may be leading to an increase in uranium concentrations in North American rivers (Spalding and Sackett 1972). Also, natural soil erosion probably contributes to the uranium load in rivers. Levels in the range of 1 to 2 micrograms/L were seen in Texas rivers. Twenty years earlier (Rona and Urey 1952), levels in other rivers in North America averaged at 0.026 micrograms/L. The ratios of uranium to phosphate seemed to indicate that some of the uranium was coming from fertilizers. It was shown that corn, soybeans and alfalfa take up very little uranium from the fertilizers. More than 99% of the uranium transported by run-off from land to freshwater systems remains with suspended particulates on the sediment. Very little uranium is in true solution in fresh water (Spalding and Sackett 1972) (see Table IV-10).

Uranium may be introduced into drinking water supplies during the mining and milling of uranium ore. This water can be treated with an ion exchange process to reduce the uranium concentration. Anion exchange was shown to produce over 90% removal at the 80-micrograms/L level (Lee et al. 1981). In

Table IV-10 Water from Uranium Mines in New Mexico

Mine	Uranium (micrograms/L)
Church Rock I & IE	1,140
UN-Old Church Rock	11,400
UN-Homestake Partners IX	2,420
Kern-McGee Western and Central Ambrosia Lake	2,440
UN Corporation IX	2,230
Kern-McGee Section 35IX	390
Ronchers-Johnny M	2,250
Anaconda P-10	4,790
Sohio JJ #11	31,500
UN Corp. St. Anthony	5,510
UN Northeast Church Rock	1,200
Gulf Mariano Lake	1,510

Adapted from Water Quality Data for Discharge from New Mexico Uranium Mines and Mills, New Mexico Health and Environment Deptartment, July, 1980.

some cases, uranium has produced a sediment concentration is settling ponds.

A survey of uranium mines in New Mexico revealed the water effluent concentrations shown in Table IV-10. As can be seen, such concentrations are an important potential source of uranium in drinking water supplies.

Accidental releases of uranium from tailings pond dams are another potential source of uranium contamination of water supplies. On July 6, 1979, a breach developed in United Nuclear Corporation's tailings pond dam near Church Rock, New Mexico. Approximately 93,000,000 gallons of tailings and liquor containing uranium was released into a tributary of the Rio Puerco River. The contamination went into the Rio Puerco River, which passes through Gallup and into the state of Arizona.

Table IV-11 shows the concentrations in the river before and after the spill. Fortunately, the poorer quality of the river water had previously prevented its use as a source of drinking water. However, the population was potentially exposed to ionizing radiation through dust that was resuspended from contaminated soils and through comsumption of contaminated vegetation and of beef and milk from cows that had consumed contaminated vegetation. The maximum risk from these sources was estimated to be 0.75×10^{-6}/year (USEPA 1980c).

Table IV-11 Uranium Concentration in Rio Puerco River at Gallup, New Mexico

Date	Concentration (pCi/L)
October 4, 1978	28.4
May 21, 1979	39.3
August 2, 1979	37.3
August 20, 1979	136.7
September 25, 1979	393.3
October 23, 1979	320.0
December 4, 1979	200.0

Adapted from USEPA 1980c.

As shown in Table IV-12 (USEPA 1980a), the concentration of uranium in the atmosphere can be as high as 200 airborne particles or more per Ci/m^3.

It is concluded that if drinking water contains uranium at a concentration of 10 pCi/L, it will represent the major (more than 90%) source for humans. Further, most of the community public water supplies with major concentrations of uranium are found in the western mountain states (see Table IV-9). Thus, the supplies affected will be the smaller population areas that use groundwater.

C. Uranium in Foods

The concentrations of uranium in most foods is in the 1 to 100 ng/g range, as shown in Table IV-13. The dietary intake varies, as shown in Table IV-14, although the overall intake is in the range of 1.3 to 1.4 micrograms/day (Hursh and Spoor 1973). Table IV-15 shows the distribution of foodstuffs in the diet by percentage and estimates an intake of 1 microgram/day (Hursh and Spoor 1973). Another estimate of daily intake of uranium in the U.S. is about 0.4 pCi/day (UNSCEAR 1977), with a range of 0.2 to 0.9 pCi/day. Concentration factors for uptake of uranium by terrestrial plants, including staple grains and market garden vegetables grown in contaminated soil, are in the order of 10^{-3}. Thus, ingestion of uranium

Table IV-12 Uranium in Airborne Particulates[a]

	^{234}U		^{235}U		^{238}U	
Location	aCi/m^3	± e	aCi/m^3	± e	aCi/m^3	± e
AL: Montgomery	8.5	1.3	0.5	0.3	7.5	1.2
CA: Berkeley	8.3	1.1	1.1	0.4	9.1	1.2
CA: Los Angeles	34.7	2.1	3.5	0.5	34.5	2.1
CO: Denver	65.3	3.9	9.4	1.0	61.5	3.7
FL: Miami	17.1	1.8	2.1	0.5	12.6	1.4
ID: Idaho Falls	51.2	3.0	5.1	0.6	59.0	3.3
IL: Chicago	35.1	2.1	2.7	0.4	36.1	2.1
ND: Bismarck	48.4	3.5	5.0	0.8	45.2	3.3
NJ: Trenton	16.6	2.3	2.4	0.8	17.6	2.4
NM: Santa Fe	62.1	6.1	3.8	1.0	75.8	9.4
NV: Las Vegas	120.0	6.4	12.2	1.1	81.8	4.6
NY: New York	17.8	2.1	1.1	0.4	16.5	2.1
NY: Niagara Falls	111.0	10.0	6.3	1.4	122.0	10.9
OH: Columbus	63.2	5.0	6.8	1.1	53.9	4.4
OK: Oklahoma City	72.5	7.6	9.4	2.5	73.9	7.7
OR: Portland	17.6	1.5	7.3	0.9	22.7	1.8
PA: Harrisburg	19.8	1.4	3.5	0.5	20.0	1.4
PA: Pittsburgh	75.6	5.3	9.5	1.2	81.1	5.6
SC: Barnwell	24.4	2.0	3.5	0.7	22.8	1.9
SC: Columbia	57.6	3.0	3.3	0.5	50.1	2.7

[a] April to June 1978 Composites.

Table IV-12 - (continued)

	^{234}U		^{235}U		^{238}U	
Location	aCi/m^3	± e	aCi/m^3	± e	aCi/m^3	± e
VA: Lynchburg	73.6	4.0	5.5	0.6	25.0	1.7

Adapted from USEPA (1980a).

Table IV-13 Concentrations of Uranium in Some Foods

Food	U concentration (ng/g raw weight)	Reference
Oats, wheat, millet and peas	10-100	a
Potatoes	15	a
	18	a
Carrots	7.7	a
Cabbage	4.7	a
Meat	20	a
Beef	14	b
Beef liver	26	b
Beef kidney	70	b
Eggs	9.6	a
Cow milk	4	b
Human milk	2	a
	1	b
River fish	11	b
Welsh onion	69	b
Wheat bread	19	b
Baked products	12	a
Polished rice	15	b
Tea	5	c
Coffee	6	c
Parsley	60	c
Red pepper	5	c
Tonagor	30	c
Mustard	0.2	c
Table salt	40	c

[a] Hursh and Spoor (1973).
[b] Novikov and Abramova (1969).
[c] Novikov (1967).

Table IV-14 Uranium Content in Foodstuffs and Annual Dietary Intake by Human Adults

	Intake (micrograms U/year)		
Product	New York	Chicago	San Francisco
Bakery and whole grain products	82	74	74
Meat and poultry	111	101	101
Flour, macaroni and rice	30	33.4	51.6
Vegetables (fresh and root)	37	33.4	56
Fresh fruit	76	76	42
Milk	16	31	62
Fish; shellfish	12.9	35.8	35.5
Eggs	3.4	3.4	3.4
Dried beans	4.5	11	11
Canned fruit; canned vegetables	8.2	8.4	8.0
Fruit juices	1.1	1.1	3.4
Total	463.8	521.9	462.5

Adapted from Hursh and Spoor (1973).

Table IV-15 The Concentration of Uranium in Various Items of Diet with Estimates of Annual Intake

Foodstuff	Mean concentration (ng U/g)	Annual uranium intake (micrograms)	Fraction of annual intake (%)
Cereals; wheat flour biscuits, spaghetti, oats and rice	0.5	68	19
Vegetables, potatoes, parsnips, turnips and sweet potatoes	1.0	138	38
Sugar	0.2	6	2
Vegetables and fruit; cabbages, sprouts, carrots and apples	0.8	92	26
Meat	0.4	16	4
Eggs	0.4	3	0.9
Fish	0.2	3	0.8
Milk	0.01	2	0.5
Fats and oils	2.0	32	9.0
Total		360	100

Adapted from Hursh and Spoor (1973).

from food is not a significant exposure pathway (Spalding and Sackett 1972).

Other possible sources of ingested uranium, such as those affecting people living near a uranium mine or mill, would represent a special case. These people could either inhale or ingest uranium as a dust or particulate or in solution.

When put into solution, uranium forms the uranyl ion (UO_2^{2+}), and this ion forms soluble salts with all common anions except phosphate (Longane 1966). This ion most commonly complexes with bicarbonate (HCO_3^-) and carbonate (CO_3^{2-}), (Yamamoto 1973) fluoride and phosphates (Katz 1951).

V. HEALTH EFFECTS IN ANIMALS

A. General

Some studies have been made of the effect of uranium on animals. Although it is not clear that the same effects will occur at the same dose levels for humans, an analysis of the animal studies can lend credence to the information on human studies. For regulatory purposes, the courts allow the extrapolation of the results of animal studies to humans.

Due to the large variation in effects for different species, it would seem difficult to extrapolate these dose values to humans. It can be concluded from animal studies (Yuile 1973), however, that chemical compound, age and species are critically important. It can also be concluded that small ingested doses of uranium produce changes not only in the kidney, but also in the cardiovascular, endocrine, hematopoietic and immune systems, as well as liver. The LD_{50} of natural uranium is sex and strain dependent and varies from 6 to 25 mg/kg (Durbin and Wrenn 1975).

Recent experiments by Pelayo et al. (1983) suggest that the immature dog kidney is more tolerant to the nephrotoxic effects of uranium than is the kidney of a more mature animal. Intravenous doses of 10 mg uranyl nitrate/kg body weight were administered to groups of puppies 1 to 2 weeks and 3 to 5 weeks of age. A marked difference in kidney functional change was noted, with the younger animals being much less affected. The same difference in tolerance was confirmed by histopathological examination of these animals.

Different uranium compounds had pronounced effects in chronic and acute toxicity. For instance, the dietary percentages that produced equal weight depression in animals were Yuile, 1973):

UO_2F_2	0.25%
Uranyl nitrate	1.0%
UF_4	20%

At 2% uranyl nitrate, the renal tubules were moderately injured, but they recovered.

Morrow et al. (1982) administered UO_2F_2 and UF_6 intravenously to beagle dogs to compare toxicity in the presence or absence of HF (the decomposition product of the former compound). They found that an absorbed dose of 10 micrograms U^6/kg body weight was effective in producing renal damage. Furthermore, a kidney concentration of 0.3 micrograms/g body weight appeared to be the threshold for renal injury.

The one general conclusion from animal studies is that the predominant effect of uranium is to cause necrotic changes in the proximal convoluted tubules of the kidney (Yuile 1973).

However, not all the studies found lesions in animals similar to those seen in human nephritis.

Recent work by Haley (1982) has detailed the pars recta of the proximal tubule as the specific site of uranium toxicity in the rat kidney. She also found that damage from uranyl nitrate in drinking water was unaffected by the addition of saline -- a protective measure against poisoning suggested for other heavy metals.

B. Chemical Toxicity

1. Mortality or Poisoning Studies

The first known description of biological effects of uranium was by G. C. Gmelin in 1824 (Hodge 1973). This was part of a study of the poisonous effects of metals on animals such as dogs and rabbits. Uranyl salts were given by mouth and shown to be a feeble poison. The effect noted was mainly irritability of the heart and coagulation of the blood. Thirty years later, in 1853-54, Leconte showed that ingestion of uranium as the nitrate led to glycosuria in dogs and rabbits (Hodge 1973). He guessed that the uranyl nitrate directly affected the kidney and used it to induce experimental nephritis. The lethal dose range for these animals was shown to be in the range of 0.5 to 1 g.

Blake, in 1868, while studying the effect of uranium nitrate on animals, found adverse effects after administering doses ranging from 4 mg up to 350 mg (Hodge 1973). Within one to two weeks, the animals died with stomach and duodenal damage. No apparent kidney problems were seen.

Table V-1 lists animal studies of the toxicity of uranium compounds from their beginning in 1825 up to the 1930's. By the turn of the century, it was apparent that the kidney was the main affected organ, with nephritis being caused by doses down to about the 1-mg/kg level (Hodge 1973). During this period, there were some other warnings of toxicity, during this period mainly relating to the G.I. tract.

Kidney damage from uranium poisoning is so predictable that it is commonly used as a model by biomedical researchers studying renal failure. Recent examples of such work dealt with rats (Goel et al. 1980, Avasthi et al. 1980, Mendelsohn and Smith 1980, Braunlich and Fleck 1981), rabbits (Ghadially et al. 1982) and dogs (Szocs et al. 1979, Tyrakowski 1979a,b, Nizet 1981). In all such cases, the doses used were relatively high, and the route of administration was typically by intravenous injection, where variable absorption is not a factor.

Studies of the effects of uranium on other organs and tissues reveal a lower degree of toxicity than is found for kidneys. Goel et al. (1979) fed 100 mg/kg uranyl nitrate to rats every other day for nearly a month. They found quite measurable histopathological damage in the liver. Jadon and Mathur (1983) observed decreased testis weight and damage to the seminiferous tubules after seven days following daily i.p. injections of uranyl nitrate in mice. Malenchenko et al. (1978) found damage to the testes and also to the thyroid in rats dosed with uranyl nitrate. They carried out both acute and chronic studies (drinking water containing 0.1% uranyl nitrate for four months).

Many studies have been done on animals using an exposure route other than ingestion. These studies (Luckey et al. 1975, Haven and Hodge 1949) show LD_{50}s and LDs in the range of 0.21 mg/kg to 1,400 mg/kg, as shown in Table V-2. Recently, deRey et al. (1984) reported that even highly insoluble uranium dioxide produced toxic symptoms following subcutaneous implantation.

The following are the lethal acute doses of uranyl nitrate administered intravenously (a one-month study) (Eve 1964): rabbit, 0.1 mg/kg; guinea pig, 0.3 mg/kg; rat, 1 mg/kg and mouse, 10 to 20 mg/kg.

While most studies have involved laboratory animals, a recent project used wild ducks to assay uranium toxicity (Haseltine and Sileo 1983). Dietary doses of up to 1,600 ppm powdered uranium were fed in the ducks' mash for six weeks. Neither histological nor physiological damage was observed.

Table V-1 Toxicity of Uranium Compounds for a Single Exposure

Compound	Animal	Route	Toxic dose	Dosage (expressed as mg of compound)
Uranyl nitrate $[UO_2(NO_3 - 6H_2O]$	Dog	oral	LD	1400
	Dog	s.c.	LD_{50}	14
	Dog	i.v.	LD	6.75
	Mouse	i.p.	LD_{50}	50
	Mouse	i.v.	LD	30
	Rat	i.p.	LD_{50}	305
	Rat	i.p.	LD_{50}	135
	Rat	i.v.	LD	2.11
	Guinea pig	i.v.	LD	0.63
	Rabbit	i.v.	LD	0.21

Adapted from Luckey et al. (1975).

The most extensive studies of chronic oral toxicity of uranium on animals were done during and after World War II. These studies from the Manhattan Project (Metallurgical Laboratory) and the University of Rochester have been summarized by Yuile (1973). The studies were done for 30 days, one year and two years with nine different compounds (only four compounds for the longer studies) on mice, rats, dogs and rabbits. These studies involved large doses, and although they are of interest in indicating effects to look for at lower doses, they do not necessarily indicate what the effects will be at lower doses.

One of the most important results of the 30-day study was that different chemical forms of uranium resulted in considerable differences in effect. It was found that UO_2, U_3O_8 and UF_4, which are only somewhat soluble, can be considered to be non-toxic. Diets with up to 20% of these compounds for 30 days caused no mortality and little, if any, interference in growth. For the soluble compounds (UO_3, $UO_2(NO_3){\cdot}6\ H_2O$, UO_2Ac_2, UO_4), however, results were quite different. Diets of 2% to 10% for 30 days led to 100% mortality, and diets in the range of 0.1% to 1% caused growth depression. All rabbits receiving 0.5% died, while none at 0.02% died. However, histological renal damage was observed at all levels.

For the one- and two-year studies at the Metallurgical Laboratory (Yuile 1973), large groups of male and female rats were fed both soluble compounds (UO_2F_2 and uranyl nitrate) and insoluble compounds (UF_4 and UO_2). The minimum percentage in the diet which produced growth depression was 25% UF_4. No growth depression was seen for UO_2.

Novikov and Yudina (1970) have reported on year-long studies in rabbits and rats of uranium toxicity from drinking water. They used uranyl nitrate doses of up to 60 mg/L for the rats, but found rather inconclusive results with regard to kidney and liver damage.

Even a single moderate dose of uranium can have long-term effects on kidney structure and function. Haley et al. (1982) injected rats with 10 mg uranyl nitrate/kg body weight and observed histopathological effects for eight weeks thereafter. Most early functional changes were reversed before the end of the experimental period.

2. Low-Level Exposure

In a study (Yuile 1973) of 30 dogs fed $UO_2(NO_3)_2 \cdot 6\ H_2O$, UF_4, UCI_4 or UO_2F_2 for one year at doses of 0.0002 to 10 g/kg, adverse effects on growth were noted only for those dogs receiving 0.2 g/kg of uranyl nitrate in the diet. The smaller concentrations were used for the soluble compounds, such as UO_2F_2, while the insoluble compounds, such as UO_2, required larger concentrations to produce an effect. A few animals receiving the highest dose showed some renal cortical tubule damage.

Chronic toxicity may also be related to enzyme poisoning in the kidneys (Leussenhop et al. 1958), along with some liver damage as a result of the kidney damage (Venugopal and Luckey 1978). Experiments with animals which inhaled uranium compounds for a year showed mild kidney changes associated with kidney burdens of about 1 microgram uranium/g.

Studies with an average dose of 0.1 to 0.2 mg/kg/day to dogs produced the following effects: fall in creatine clearance (90% ± 6% decrease), rise in plasma urea nitrogen, proteinuria, aminoaciduria and weight loss (Fukuda and Kopple 1980, Nomiyama and Foulkes 1968).

Damage to the kidneys from uranium is thought to be the source of alkaline phosphatase entering the blood. This effect has been seen in rabbits with doses as low as 1 mg/kg. This dose level also produces inhibited metabolism of nucleic acids in the kidneys and liver. In an 11-month study of albino rats, the following data were produced (Novikov 1970):

U Concentration (mg/L)	Phosphatase activity in blood serum
0.05	13.8 ± 3.3
0.6	15.4 ± 4.3
6	13.3 ± 1.1
60	26.2 ± 4.2

Thus, above 6 mg/L, a definite effect can be seen. Below that level, it is not clear whether any effect is present. This study and the follow-up (Novikov 1972) involved 42 rabbits, 230 white rats and 32 guinea pigs. The rabbits and guinea pigs were fed uranyl nitrate by mouth at 0.02, 0.2, 1.0 and 2.0 mg/L, and the rats and guinea pigs were fed at doses of 0.05, 0.6, 6 and 60 mg/L. Morphological changes were found in the kidney tubules at 60 mg/L, but no mention was made of looking for this result at lower dose levels.

Another Russian study (reports for this study and that discussed above suffer somewhat in translation) involves a chronic exposure (Gus'kova et al. 1966). It was a two-year study involving 500 albino male rats. They were gavaged uranyl nitrate per os with a metal tube at dose rates of 0.016, 0.08 and 16 mg/kg/day. No changes in blood nitrogen were noted. Urinary creatine and creatinine were used as indices characterizing disorders in the reabsorptive capacity of the kidneys of the rats. Only the 16-mg/kg level produced changes, and they were described as "significantly increased."

If we argue that a rat consumes 0.035 L/day and weighs 350 g, then at the 0.08-mg/kg dose level:

$$\frac{0.08\ \text{mg}}{\text{kg/day}} \times \frac{0.350\ \text{kg}}{0.035\ \text{L/day}} = 0.8\ \text{mg/L}$$

The Russian studies have several drawbacks. They involved only a few animals and dose levels. Very little or no actual data were given in the papers. The studies did not look for casts or albumin in the urine, and there was no follow-up pathology on the kidney. Since it was noted that the death rate was statistically the same in the control and experimental work, the results are essentially inconclusive.

The Canadians have recently reported (Canadian Radiation Protection Bureau 1980) the results of some chronic toxicity studies on animals. Rats were fed uranyl nitrate for 11 months in water. No significant effects were seen at a rate of 0.1 mg/kg/day. At 0.9 mg/kg/ day, an increase in serum alkaline phosphatase activity was seen. At 9 mg/kg/day, nucleic acid metabolism in kidney and liver was inhibited. Doses of 0.042 to 0.42 mg uranyl nitrate/kg/day for 12 months showed no significant effects, while a dose rate of 2.11 mg/kg/day produced a decreased activity of acid phosphatase in the spleen and inhibited the metabolism of the nucleic acids in the kidneys and liver. Dogs fed uranyl nitrate for 21 months at 0.12 mg/kg/day showed only minor hematopoietic deficiency, but at a dose rate of 2.11 mg/kg/day, the following effects were manifested: changes in blood cell morphology, disturbance of thyroid function, increased basal metabolism, changes in hepatic function and hematopoietic deficiency.

Only one group has reported kidney tumors due to ingestion of uranium (Filippova et al. 1978). They reported tumors for single doses as low as 0.55 mg/kg administered intratracheally (not by ingestion). Except for bone tumors, no other conclusive exceptions were reported.

From the animal studies, it is clear that ingested uranium leads primarily to kidney damage. It would appear that this effect occurs at dose levels as low as 1 mg/kg/day and perhaps lower. Other deleterious effects are seen at lower dose levels, but few if any occur at dose levels below 0.1 mg/kg. Because of variations in the fraction of a dose actually absorbed and present in the kidney, it is not possible to uncritically relate oral dose to extent of kidney damage. Because of major flaws in or questions about the studies, it is not clear at this time exactly what effects low-level chronic ingestion produces in animals. More work will have to be done to determine what the no-effect level is.

C. Radiotoxicity

The long-term radiotoxic effects of intravenously injected ^{232}U, ^{233}U, U_{nat} and ^{226}Ra in mice have been investigated (Finkel 1953, data of Finkel, quoted in Durbin and Wrenn 1975).

The results for uranium are shown in Table IV-3. At high doses, all of the nuclides reduced the life span. It was concluded that the biological effectiveness of ^{232}U, ^{233}U and ^{226}Ra are similar. The data fit linear dose-response curves. The slopes are:

Injection	Percent bone tumors per rat
^{232}U	0.016
^{233}U	0.012
^{226}Ra	0.12

In a later study, the ^{226}Ra slope was 0.043% bone tumors/rat. It is clear from the Finkel study that ingestion of certain uranium isotopes leads to bone cancers in a way that is both qualitatively and quantitatively similar to the results from ingestion of radium. Neither bone cancer nor marrow damage, however, has been reported for animals receiving only natural uranium.

Table V-2 Longevity and Number of Animals with Malignant Bone Tumors After Intravenous Injection of Uranium Isotopes Into Adult CF1 Female Mice

Dose		Animals			Lifespan		No. of mice with malignant bone tumors
Activity (Ci/kg)	Mass (mg/kg)	No. Mice injected	No. of survivors 30 day	No. of survivors 210 day	Median (days)	Max. (days)	
^{232}U							
10	4.6×10^{-4}	30	30	30	426	498	18
1	4.6×10^{-5}	45	45	43	510	719	5
0.1	4.6×10^{-6}	60	60	60	478	775	2
^{233}U							
100	10	15	0[a]	0	2	6	0
75	7.5	15	0[a]	0	4	6	0
50	5.0	30	12[b]	0	17	181	0
25	2.5	45	36[a]	32	321	632	10
10	1.0	60	56[a]	54	450	675	13
5	0.5	75	70[b]	67	440	687	9
2.5	0.25	90	90	88	446	709	7
1.0	0.1	105	105	95	457	743	5
0.5	0.05	120	120	117	468	763	3
0.1	0.01	120	120	117	448	695	1
U_{nat}							
6.8×10^{-4}	1	30	30	30	468	629	0
6.8×10^{-5}	0.1	45	45	43	460	699	0
6.8×10^{-6}	0.01	60	60	56	424	734	0

[a] Deaths all occurred in first seven days.
[b] Two-thirds of deaths occurred in first seven days.

VI. HEALTH EFFECTS IN HUMANS

Homeopathic physicians searching for beneficial effects first tried uranium on humans in 1860. It was thought for several years that the nitrates of uranium could be used to treat diabetes mellitus and chronic albuminuria. In 1880, the recommended dose of UNO_3 for treatment of diabetes mellitus was 10 to 20 mg t.i.d. (three times a day). One of the recommended mixtures was Vin Urane, which was one part UNO_3, 50 parts glycerol and 1,000 parts red wine. Although there was never any good evidence that UNO_3 had a beneficial effect, its use was mentioned in the literature up to the 1930's. This history has been thoroughly traced by Hodge (1973).

All available information relating to the oral toxicity of uranium to humans involves acute exposures (Hursh and Spoor 1973). No experimental studies have been conducted concerning the chronic low-level oral toxicity of uranium to humans, but epidemiological studies do exist. These studies (e.g., Novikov and Abramova 1969), however, do not show any adverse effects from uranium exposure. Information is available concerning the chronic toxicity of other bone seeking elements, such as radium in the watch dial painters (Rowland et al. 1978), and that information may be helpful in setting a standard for uranium.

The acute studies are of some value in that they demonstrate characteristic symptoms and give some information regarding uptakes.

Two studies involving the intravenous injection of uranium into terminal patients reveal some useful information (see Table VI-1). In one study, ^{235}U was to be used in neutron capture therapy of brain tumors (Luessenhop et al. 1958). The therapy involved introducing a substance (^{235}U in this case) that concentrated in the tumor. After neutron irradiation, the decay energy of the fission products of ^{235}U would destroy the tumor. Before conducting the therapy, the tolerance to intravenous uranium was determined. The study involved five patients, whose life spans and amounts of uranium received are shown in Table VI-1. All of the patients were in a coma or semi-coma until their demise.

The study involved collecting blood, urine, feces and bone. The symptoms produced included some evidence of epithelial casts, increased urinary output, decreased serum sodium and serum chloride and increased serum potassium. There was initial damage to the distal portion of the proximal convoluted tubule of the kidney; this elevated the concentration of

Table VI-1 Data for Patients Treated for Brain Tumors with Uranium and Neutron Irradiation

Patient	Uranium nitrate (mg/kg)	Duration of life after injection (days)
1	0.00	2.4
2	0.13	60
3	0.07	566
4	0.17	42
5	0.28	68

From Luessenhop et al. (1958).

uranyl cations next to the epithelium of the proximal convoluted tubule, viz., bicarbonate, chloride, glucose and amino acids.

The excretion of albumin and the enzyme catalase in the urine was seen, reaching a maximum in three to six days and then returning to normal. The earliest observed sign of renal tubular damage was an increase in urinary catalase and albuminuria. The double peaking observed for both catalase and albumin was due to an increase in the membrane permeability of the tubular walls, followed by the breakdown of the cell structure. (Increased catalase excretion has been seen in rabbits and dogs at the dose level of 0.005 mg U/kg.) The study also noted casts, and along with the tubular damage, interference with the renal capacity for re-absorption of sodium and chlorine and secretion of potassium was indicated. This study concluded that the lethal dose for acute injection of uranium in humans was about 1 mg/kg (i.e., about 60 mg in the bloodstream would produce a human fatality). About 0.1 mg/kg was the minimal dose necessary to produce catalasuria and albuminuria.

Nechay et al. (1979) carried out *in vitro* studies of the inhibitory effect of uranyl nitrate on the adenosine triphosphatase enzymes of a variety of human and mammalian tissues. They found I_{50} values (amount of substance inhibiting enzyme activity to 50% of normal) for uranyl nitrate with human kidney homogenates of 1 to 2 x 10^{-4} M. These were in the same order of magnitude as values for dogs, cats, hamsters, rabbits and rats, which lends credence to the practice of extrapolating experimental results obtained with animals to humans.

Another study (Bernard 1958) of acute injection of uranium involved patients in the terminal stage of an irreversible central nervous system disease. The doses injected ranged from 4 to 50 mg. The study showed that the critical organs were the kidneys and skeleton and that the "safe burden" to the kidney was one-tenth that allowed on radiological considerations.

Only a few studies have been done on oral ingestion of uranium, and all involved acute doses (Hursh and Spoor 1973). In one case, the uranium was being used as a "therapeutic" agent in doses of 30 to 60 mg of uranyl nitrate given two to three times a day. The symptoms manifested were sugar in urine, reduced volume of urine and thirst. After treatment, the patients returned to their original status. In another case, a volunteer ingested 1 g of uranyl nitrate and showed diarrhea, violent vomiting and slight albuminuria. It was roughly estimated that 1% was absorbed from the gut.

A study was made in Russia for two towns with different uranium concentrations: 40 to 50 micrograms/L and 2 to 4 micrograms/L (Novikov 1967, Novikov et al. 1968). The towns had populations of 5,764 and 3,303, respectively. No differences were seen in the birth and death rates of these towns. Furthermore, there were no differences in the incidence of malignant neoplasms. Specifically, deaths due to bone cancers and leukemias were apparently considered, but no differences were found. This result would not be surprising if the risk of such deaths is on the order of 10^{-5}/lifetime, as calculated in Section VIII.

No information exists describing the long-term effects (e.g., bone cancer) of chronic low-level ingestion of uranium by humans (NAS 1980a). An estimate can be obtained, however, by converting the slope from the linear fit to the dose-response data for mice generated earlier, assuming that the mouse data are pertinent to man. The slope for humans, B_H, is related to that for mice, B_M, by the body weights in the relationship:

$$B_H \quad = \quad B_M(W_H/W_M)^{1/3}$$

where W_H = 70 kg for humans and W_M = 0.03 kg for mice. Then:

$$\begin{aligned} B_H \quad &= \quad 0.015 \text{ bone tumors/rad} = (70 \text{ kg}/0.03 \text{ kg})^{1/3} \\ &= \quad 0.20\% \text{ bone tumors/rad} \end{aligned}$$

Since the mice data were for a lifetime, we can take the value above to be a lifetime risk. Thus, the value is 2×10^{-3} bone tumors/rad/person/lifetime. Since 20 rem = 1 rad, for 100 mrem the risk is:

$$\frac{2 \times 10^{-3} \text{ bone tumors}}{\text{rad/person/lifetime}} \times \left[\frac{\text{rad}}{20 \text{ rem}}\right] \times \left[1 \text{ rem}\right]$$

$$= 10^{-5} \text{ bone tumors/person/lifetime}$$

This is consistent with the risk value calculated in Section VIII.

Skeletal alpha radiation doses from uranium can be calculated from measured equilibrium concentrations of skeletal uranium. Most such measurements have come from the United States or other industrialized nations. Fisenne et al. (1983) recently made careful measurements of uranium concentrations in human bones from Nepal and Australia. From these, they calculated the annual alpha doses to bone from ^{238}U to be 0.039 mrad for the Nepalese and 0.009 mrad for the Australians. Such wide variations are not unexpected because of the known geographic differences in uranium concentration in human bone.

Alternatively, the risk of bone cancer induction from uranium can be related to that due to radium using data for ^{226}Ra. Both are rather uniformly distributed throughout the volume of mineral bone. Using an average daily ingestion of 5 pCi of uranium, the calculated lifetime risk of nine bone sarcomas per million people ingesting 5 pCi ^{226}Ra/day (Cothern, 1983), and the equilibrium skeletal burdens and summed alpha decay energies for the two nuclides, they arrived at the following equation:

$$\begin{array}{l}\text{Lifetime risk} \\ \text{(long-lived U)}\end{array} = \frac{[9 \text{ bone sar.}]}{[10^6 \text{ persons}]} \times \frac{[55 \text{ pCi U in skel.}]}{[125 \text{ pCi } ^{226}\text{Ra in skel.}]} \times \frac{[4.5 \text{ MeV from U}]}{[12.11 \text{ MeV } ^{226}\text{Ra + dau.}]} = \frac{1.5 \text{ bone sarcomas}}{10^6 \text{ persons}}$$

Because of assumptions concerning the ratio of skeletal content to the daily ingestion of uranium, the predicted bone sarcomas would actually range from 0.1 to 5 per million persons. This should be contrasted to about 750 naturally occurring bone sarcomas per million persons.

This predicted number of bone sarcomas induced by ingestion of natural uranium is based on a linear dose-response model. The nature of and basis for such a model have been extensively discussed in a recent report by the National Academy of Sciences (1980b). If the alternative dose-squared model is correct, however, Mays et al. (1985) predict that virtually no cancers would be expected from environmental levels of uranium.

Spoor and Hursh (1973, p. 250) have stated, "The implication that the contamination of drinking water by uranium is an uncommon and relatively unimportant hazard is confirmed by the dearth of precise and unambiguous information in the literature." This conclusion still seems pertinent. The total absence of any data showing radiological toxicity for natural uranium in humans or laboratory animals suggests that only chemical toxicity should be considered for human safety (NAS 1980a, Sciences 1980a, Wrenn et al., 1985).

There is currently some uncertainty as to what constitutes a "safe" level of uranium in drinking water from the standpoint of chemical toxicity. In 1973, Hursh and Spoor were willing to accept about 600 pCi/L. The U.S. Environmental Protection Agency (Cothern et al. 1983) has recently issued a health effects guidance level of 10 pCi/L, based somewhat on recommendations of a National Academy of Sciences report (1980a). Most recently, Wrenn et al. (in press) recommended a level of 67 pCi/L. All of these levels are orders of magnitude below the no-effect level seen in animal studies.

VII. MECHANISMS OF TOXICITY

Uranium has been recognized as a chemically toxic element since the middle of the 19th century. Its potential for being radiotoxic, as well, only became evident at the turn of the present century. Hodge (1973) has provided a comprehensive history of uranium poisoning from the time of its discovery until World War II. The emphasis here will be on chemical toxicity, since radiotoxicity has not been observed with natural uranium in either man or animals (Wrenn et al., in press).

A. Chemical Toxicity

The main chemically toxic effect of uranium is on the kidneys. This has been evident for over a century from both medical administration to humans and numerous animal studies. Nephritis (inflammation of the kidneys) and changes in urine composition are the clear symptoms. Thus, both kidney structure and function are affected.

Yuile (1973) has reviewed these effects on the kidney in great detail. Morphologically, the most evident changes are in the proximal convoluted tubules of the nephron units. Necrosis of the tubular lining cells occurs first, followed by a clogging of the tubules with cellular debris and the appearance of this debris (casts) in the urine. In non-fatal cases, the tubular lining cells are regenerated within two to three weeks, but differ in appearance from normal cells. This dramatic damage tends to overshadow the more subtle damage to the glomerulus of the nephron. The glomerulus is the filtering cup of the nephron which encloses a tuft of capillaries. Damage here may account for the appearance of protein in the urine following uranium exposure.

The cellular and biochemical mechanisms that bring about nephritis have been generally understood for several decades (Dounce 1949, Yuile 1973, Durbin and Wrenn 1975). Uranium (VI) is transported in the blood complexed with serum proteins and bicarbonate ion. The U(VI)-bicarbonate complex filters through the glomerulus into the nephron tubule, where three actions result in uranium deposition. The bicarbonate is reabsorbed through the tubule lining, the pH decreases, and water is reabsorbed, concentrating the uranium solution. Thus, uranyl ion (UO_2^{++}) complexes with proteins on the surfaces of the cells lining the tubule, eventually causing their damage and death. This is apparently a physiochemical change, not an enzyme poisoning, since there is a lag period before damage is evident and the complexing is reversible. Damage to the lining of the proximal tubules leads to the commonly observed increase in excretion of glucose, certain amino acids and some enzymes.

In a variety of animals, either higher acute or lower chronic doses of soluble uranium compounds may result in irreversible damage to a critical mass of proximal renal tubule cells, leading to death. Animals receiving sublethal doses replaced the damaged tubule cells and survive. However, the replacement cells are not structurally identical to those they have replaced and are not likely to be functionally the same, either (Wrenn et al., in press).

Bone is the other major organ of deposition for uranium on a long-term basis. No experimental data have shown any chemically toxic effect of a uranium burden on bone. Soft tissues other than kidney usually do not attain sufficiently high concentrations of uranium following oral intake to exhibit symptoms of chemical toxicity.

B. Radiotoxicity

The bone seeking characteristic of uranium suggests that carcinogenesis may result from chronic local irradiation of bone or marrow cells due to the radioactive decay of uranium or its daughters. Extensive studies using natural uranium, however, have failed to demonstrate such an effect. Bone cancers have only been seen when high specific activities of other uranium isotopes - ^{232}U and ^{233}U - have been administered (Ballou 1983). The presumption is that a bone concentration from chronic irradiation that was high enough to produce bone cancer would first cause such an elevated kidney concentration that death would occur from chemical toxicity.

In summary, chemical toxicity from uranium results in well understood and quantified kidney damage. Radiotoxicity from natural uranium is only hypothetical, never having been demonstrated experimentally.

VIII. QUANTIFICATION OF TOXICOLOGICAL EFFECTS

The quantification of toxicological effects of a chemical consists of an assessment of the non-carcinogenic and carcinogenic effects. In the quantification of non-carcinogenic effects, an Acceptable Daily Intake (ADI) is calculated. An Adjusted Acceptable Daily Intake (AADI) and Health Advisory (HA) values for the chemical are then calculated to define the appropriate drinking water concentrations to limit human exposure. For ingestion data, this approach is illustrated as follows:

$$\text{ADI} = \frac{(\text{NOAEL or LOAEL in mg/kg/day})(\text{Body Weight in kg})}{\text{Uncertainty/Safety Factor}} = \text{mg/day}$$

$$\text{AADI} = \frac{\text{ADI}}{\text{Drinking Water Volume in L/day}} = \text{mg/L}$$

where:

NOAEL = no-observed-adverse-effect level.

LOAEL = lowest-observed-adverse-effect level.

Body weight = 70 kg for adult or 10 kg for child.

Drinking water volume = 2 L per day for adults or 1 L per day for children.

Uncertainty/Safety Factor = 10, 100 or 1,000.

Utilizing these equations, the following drinking water concentrations are developed for non-carcinogenic effects:

1. A one-day HA for 10-kg child.
2. A one-day HA for 70-kg adult.

3. A ten-day HA for 10-kg child.
4. A ten-day HA for 70-kg adult.
5. A lifetime AADI for a 70-kg adult.

The distinctions made between the HA calculations (items 1 through 4) are associated with the duration of anticipated exposure. Items 1 and 2 assume a single acute exposure to the chemical. Items 3 and 4 assume a limited period of exposure (possibly 1 to 2 weeks). The HA values will not be used in establishing a drinking water standard for the chemical. Rather, they will be used as informal scientific guidance to municipalities and other organizations when emergency spills or contamination situations occur. The AADI value (item 5) is intended to provide the scientific basis for establishing a drinking water standard based upon non-carcinogenic effects.

A NOAEL or LOAEL is determined from animal toxicity data or human effects data. For animal data, this level is divided by an uncertainty factor because there is no universally acceptable quantitative method to extrapolate from animals to humans. The possibility must be considered that humans are more sensitive to the toxic effects of chemicals than are animals. For human data, an uncertainty factor is also used to account for the heterogeneity of the human population in which persons exhibit differing sensitivity to toxic chemicals. An Office of Drinking Water (ODW) modification of the guidelines set forth by the National Academy of Sciences (NAS 1977,

1980) is typically used in establishing uncertainty factors as follows:

- An uncertainty factor of 10 is used when good acute or chronic human exposure data are available and are supported by acute or chronic toxicity data in other species.

- An uncertainty factor of 100 is used either when good acute or chronic toxicity data identifying NOEL/NOAEL are available for one or more species (but human data are not available), or when good acute or chronic toxicity data identifying a LOAEL in humans are available.

- An uncertainty factor of 1,000 is used either when limited or incomplete acute or chronic toxicity data are available, or when only the acute or chronic toxicity data identify a LOAEL (but not NOEL/NOAEL) for one or more species.

The uncertainty factor used for a specific risk assessment is judgmental. Factors that cannot be incorporated in the NAS/ODW guidelines for selection of an uncertainty factor, but must be considered include: (1) the quality of the toxicology data, (2) the significance of the adverse effects and (3) the existence of counterbalancing beneficial effects.

If toxicological evidence requires the chemical to be classified as a potential carcinogen (there is sufficient evidence to classify uranium as a carcinogen in humans following ingestion), mathematical models are used to calculate the estimated excess cancer risks associated with the ingestion of the chemical via drinking water. The bioassay data used in these estimates are from animal experiments. In order to predict the risk for humans, these data must be converted to an equivalent human dose. This conversion includes correction for non-continuous animal feeding, non-lifetime studies and difference in size. The factor that compensates for the size difference is the cube root of the ratio of the animal and human body weights. It is assumed that the average human body weight is 70 kg and that the average human consumes 2 L of water per day. The multi-stage model is then fit to the equivalent human data to estimate the risk at low doses. The upper 95% confidence limit of this estimate is used. Excess cancer risks can also be estimated using other models such as the one-hit model, the Weibull model, the logit model and the probit model. There is no basis in the current understanding of the biological mechanisms involved in cancer to choose among these models. The estimates of low doses for these models can differ by several orders of magnitude.

The scientific data base used to calculate and support the setting of risk rate levels has an inherent uncertainty. This is because the tools of scientific measurement, by their very nature, involve both systematic and random error. In most cases, only studies using experimental animals have been performed. There is thus uncertainty when the data are extrapolated to humans. When developing risk rate levels, several other areas of uncertainty exist, such as (1) incomplete knowledge concerning the health effects of contaminants in drinking water, (2) the impact of test animal age, sex and species and the nature of target organ systems examined on the toxicity study results and (3) the actual rate of exposure of internal targets in test animals or humans. Dose-response data are usually only available for high levels of exposure, not for the lower levels of exposure for which a standard is being set. When there is exposure to more than one contaminant, additional uncertainty results from a lack of information about possible synergistic or antagonistic effects.

A. Non-Carcinogenic Effects

The main or primary chemical effect of uranium on animals appears to be to the kidney (Hursh and Spoor 1973). The study of nephritis (inflammation of the kidney) induced by UNO_3 in rabbits, mice and rats revealed structural and functional renal changes. These studies involve both acute and chronic exposures but are all at high levels compared to environmental exposures.

The pattern of uranium poisoning is described by the following sequence of maladies: polyuria, albuminuria, casts, glycosuria, oliguria and sometimes anuria, ending in either uremic death or recovery.

Two types of lesions can be observed in the kidney: (1) glomerular or vascular which can be caused by arsenic and (2) tubular or epithelial which can be caused by uranium, chromium and mercury. If the tubular epithelium is damaged, it has the ability to regenerate itself completely in two to three weeks. The new tubular lining, however, differs both morphologically and histochemically from normal epithelium. After exposure is stopped, the morphology of the regenerated epithelium will return to normal (Canadian Guidelines, 1980). There is some evidence that uranium can cause both glomerular and tubular lesions.

As shown in Table I-2 (Appendix I), there are reports of edema in rabbits with uranium nephritis for effects down to the dose level of about 1 mg/kg, and none at lower dose levels. However none of these studies involve ingestion and thus their value is in question. Recently the Canadians (Guidelines, 1980) conducted bioassay studies with mammals. Although the details of this work are not published, the available information is shown below:

> "The chronic toxicity of uranium has been investigated in animals. Rats fet uranyl for 11 months at the rate of 0.1 mg/kg/day did not

> manifest any significant effects; at the rate of 0.9 mg/kg/day for 11 months, an increase in serum alkaline phosphatase activity was noted; and at 9 mg/kg/day for 11 months, nucleic acid metabolism in the kidneys and liver was inhibited, and serum alkaline phosphatase increased. Rabbits ingesting daily 0.042 to 0.42 mg of uranyl nitrate per kg body weight for 12 months did not exhibit significant changes, whereas as 2.11 mg/kg for 12 months, the activity of acid phosphatase in the spleen decreased , and the the metabolism of nucleic acids in the kidneys and the liver was inhibited. Dogs fed uranyl nitrate for 21 months at 2.11 mg/kg/day manifested the following: changes in blood cell morphology, disturbance of thyroid function, increased basal metabolism, changes in hepatic function, and hematopoietic deficiency; however at a dose level of 0.21 mg/kg/day for 21 months, only minor hematopoietic deficiency was observed."

Five terminal patients were injected with uranium to study its toxicity (Luessenhop, 1958). This study concluded that the lethal dose for actue injection of uranium in humans was about 1 mg/kg.

B. Quantification of Non-Carcinogenic Effects

The NOAEL for chemical effects to the kidney due to ingested uranium can be taken to be in the range of 0.1 to 1 mg/kg/day. The animal uptake is approximately 0.01, and that for humans is approximately 0.05. Uranium from drinking water contributes approximately 90% of that ingested (relative source contribution, or RSC). These values can be substituted into the following formula for the AADI:

$$\text{AADI} = \frac{(\text{NOAEL})(\text{animal } f_1)(\text{adult weight})(\text{RSC})}{(\text{safety factor})(\text{water consumption/day})(\text{human } f_1)}$$

$$= \frac{(0.1 \text{ to } 1 \text{ mg/kg/day})(0.01)(70 \text{ kg})(0.9)}{(100)(2 \text{ L/day})(0.05)}$$

= 6 to 60 micrograms/L or 4 ty 40 pCi/L (rounded off to one significant figure)

where:

the NOAEL = 0.1 to 1 mg/kg/day

animal f_1 = 0.01

adult weight = 70 kg

safety factor = 100

water consumption = 2 L/day

human f_1 = 0.05

A similar estimate by the National Academy of Sciences (1980a) yields a value of 35 micrograms/L. This estimate, however, assumes that only 10% of the ingested uranium comes from drinking water.

C. Carcinogenic Effects

There is no direct experimental evidence of the radiotoxicity of ingested natural uranium. There are studies showing the radiotixicity due to injection of short lived uranium isotopes 232 and 233 (Finkel, 1953 and Wrenn, 1975). Uranium is a known bone seeker, however, and its radiotoxicity would be expected to be similar to that of ingested radium.

Radium that is ingested by humans is either excreted or deposited in the bone. Very little radium is deposited in other organs. The human health consequence of ingested radium is primarily bone cancer. The effect on red bone marrow, in the form of leukemia, that is predicted by the dosimetric models appears to be greater by a factor of ten than that found in epidemiological studies. The risk rates predicted by dosimetric models and from epidemiological studies among radium watch dial painters, however, are in good agreement. Studies in animals of bone cancers due to ingested radium also support the findings of the epidemiology studies (Mays, 1985).

D. Quantification of Carcinogenic Effects

Using the modified ICRP 30 dosimetric model (Sullivan 1981) and weighting factors defined from BEIR III (USEPA 1984), the risk and the annual effective dose equivalent (EDE) for ingested ^{234}U and ^{238}U are tabulated in Tables VIII-1 and VIII-2.

Table VIII-1 Calculations of the Effective Dose Equivalent Rate per pCi/L for Ingestion of ^{234}U (f_1 = 0.05)

Organ	Lifetime risk/ pCi/L/ million persons exposed[a]	70-Year dose equivalent rate/pCi/L (mrem/year)[a,b]	Weighting factor[c]	Effective dose equivalent rate/pCi/L (mrem/ year)
Red bone marrow	0.44	0.20	0.16	0.032
Endosteal bone	0.14	3.12	0.009	0.028
Thyroid	0.01	0.01	0.099	0.001
Breast	0.02	0.01	0.13	0.001
Lung	0.03	0.01	0.21	0.002
Stomach	0.02	0.01	0.084	0.001
Intestine	0.07	0.08	0.039	0.003
Liver	0.01	0.01	0.085	0.001
Pancreas	0.01	0.01	0.058	0.001
Urinary tract	0.64	1.20	0.025	0.030
Other	0.02	0.01	0.11	0.001
Total	1.4			0.10

[a] Risk and dose equivalent calculations were done at the Oak Ridge National Laboratory using the modified ICRP 30 model under an EPA contract.
[b] Assuming ingestion of 2 L/day.
[c] Derived by the USEPA from NAS (1980).

From USEPA (1984).

Table VIII-2 Calculations of the Effective Dose Equivalent Rate per pCi/L for Ingestion of ^{238}U (f_1 = 0.05)

Organ	Lifetime risk/ pCi/L/ million persons exposed[a]	70-Year dose equivalent rate/pCi/L (mrem/year)[a,b]	Weighting factor[c]	Effective dose equivalent rate/pCi/L (mrem/ year)
Red bone marrow	0.46	0.21	0.16	0.034
Endosteal bone	0.12	2.71	0.009	0.024
Thyroid	0.01	0.01	0.099	0.001
Breast	0.02	0.01	0.13	0.001
Lung	0.02	0.01	0.21	0.002
Stomach	0.02	0.01	0.084	0.001
Intestine	0.06	0.07	0.039	0.003
Liver	0.01	0.01	0.085	0.001
Pancreas	0.01	0.01	0.058	0.001
Urinary tract	0.57	1.08	0.025	0.027
Other	0.02	0.01	0.11	0.001
Total	1.31			0.096

[a] Risk and dose equivalent calculations were done at the Oak Ridge National Laboratory using the modified ICRP 30 model under an EPA contract.
[b] Assuming ingestion of 2 L/day.
[c] Derived by the USEPA from NAS (1980).

From USEPA (1984).

For ^{234}U, the lifetime risk of 1.4 cases per pCi/L per million people exposed means that the 10^{-4}/lifetime risk level corresponds to a concentration of 71.4 pCi/L. Using the EDE value of 0.10 mrem/year/pCi/L, this gives 6.0 mrem/year.

For ^{238}U, the lifetime risk of 1.3 cases per pCi/L per million people exposed means that the 10^{-4}/lifetime risk level corresponds to a concentration of 76.9 pCi/L. Using the EDE value of 0.096 mrem/year/pCi/L, this gives 6.2 mrem/year.

At equilibrium, the activities of ^{234}U and ^{238}U are equal, and the contribution of ^{235}U is negligible to natural uranium. For the calculations here, it is assumed that there is equilibrium. Similar calculations are shown in Cothern et al (1983). In nature the $^{234}U/^{238}U$ activity ratio is often greater than one and this can pesent a problem if measurements are made in micrograms and need to be converted to pCi.

Allowing an uncertainty of a factor of four to five and rounding off all figures to one significant figure, the drinking water concentrations of natural uranium for different risk rates and EDEs are shown in Table VIII-3. If the standard for uranium were to be set at the same risk level as the existing MCL for radium, it would be in the range of 10 pCi/L ($f_1 = 0.2$) and 40 pCi/L ($f_1 = 0.05$) (Cothern, 1983).

Table VIII-4 shows the protective action guidance levels, using the modified ICRP 30 dosimetric model, for short-term exposures to natural uranium by ingestion. Protection action guidance levels are used for emergency and accidental exposure situations.

E. Existing Guidelines and Standards

The existing standards and guidelines are shown in Table VIII-5. As can be seen, some standards are for chemical toxicity (shown in mg/L), and some are for radiotoxicity.

Table VIII-3 Lifetime Risk Rates and Annual Effective Dose Equivalent Rate as They Relate to Concentrations of Natural Uranium in Drinking Water[a]

Lifetime individual risk rate	Effective dose equivalent rate (mrem/year)	Drinking water concentration[b] (pCi of U/L)
10^{-3}	100	700
10^{-4}	10	70
10^{-5}	1	7
10^{-6}	0.1	0.7

[a] Assuming ingestion rate of 2 L/day.

[b] Rounded off to one significant figure and using an uncertainty of a factor of four to five.

Table VIII-4 Concentration of Uranium in Drinking Water That Produces a Whole Body Dose of 500 mrem (pCi/L) (f_1 = 0.05)

	1 day	10 day	1 year
^{234}U	3×10^6	3×10^5	9×10^3
^{238}U	4×10^6	4×10^5	1×10^4

Table VIII-5 Standards or Guidelines for Uranium in Drinking Water in the World

Country	Standard or Guideline
Canada	0.02 mg/L(13 pCi/L)
Chile	600 pCi/L maximum acceptable
Hungary	7 x 10^{-6} microcuries/mL (7,000 pCi/L)
Iran	10 pCi/L
Italy	6 x 10^{-6} microcuries/mL: occupational exposure for soluble uranium (6,000 pCi/L)
Kuwait	5 pCi/L
Qatar	0.02 mg/L(13 pCi/L)
Republic of South Africa	0.6 micrograms/L: recommended (0.4 pCi/L) 4.4 micrograms/L: maximum (0.3 pCi/L)
U.S.A.[a]	Maximum for 1 day of 4 mg/L (260 pCi/L); average for 30 days not to exceed 2 mg/L (130 pCi/L) for uranium monthly effluent

[a] From Federal Register, Vol. 43, Section 440.52, July 11, 1978.

From Survey of the Uranium Task Force, Department of Health, Province of Nova Scotia, Canada (1984).

IX. REFERENCES

Adams N and Spoor NL. 1974. Kidney and bone retention functions in the human metabolism of uranium. Phys. Med. Biol. 19:460-471.

Avasthi PS, Evan AP and Hay D. 1980. Glomerular endothelial cells in uranyl nitrate-induced acute renal failure in rats. Journal of Clinical Investigation 65:121-127.

Ballou JE. 1983. Toxicity of thorium cycle nuclides. In: Pacific Northwest Laboratory Annual Report for 1982. Pacific Northwest Laboratory, Richland, WA. (PNL-4600) Pt. 1, pp. 83-85.

Berlin M and Rudell B. 1977. Uranium. In: Friberg L (Chairman). Toxicology of metals. Vol. II, EPA 600/1-77-022, pp. 454-472.

Bernard SR. 1958. Maximum permissible amounts of natural uranium in the body, air and drinking water based on human experimental data. Health Physics 1:288-305.

Bertine KK, Chan H and Turekian KK. 1970. Uranium determination in deep sea sediments and natural waters, using fission tracks. Geochim. Cosmochim. Acta. 34:641-648.

Bhat SG and Krishawamy S. 1969. Uranium and radium in Indian waters. Proc. Indian Acad. Sci., Section A, No. 1, pp. 1-17.

Braunlich H and Fleck C. 1981. Urinary enzyme excretion as an indicator of nephrotoxicity in dependence on age. Experimental Pathology 20:182-187.

Broadway JH and Strong AB. 1983. Radionuclides in human bone samples. Health Physics 45:765-768.

Broadway JH and Strong AB. 1980. Radionuclides in human bone with estimates of population dose and health impact. U.S. Environmental Protection Agency, Washington, D.C. EPA-520/5-80-006.

Bulman RA. 1980. Some aspects of the bioinorganic chemistry of the actinides. Coordination Chemistry Review 31:221-250.

Butterworth A. 1958. Human data on uranium exposure. In: Symposium on occupational health experience and practices in the uranium industry; New York, NY, October 1958. U.S.A.E.C. Health and Safety Laboratory. HASL-58. pp. 41-46.

Butterworth A. 1955. The significance and value of uranium in urine analysis. Transactions of the Association of Industrial Medical Officers 5:36-43.

Carpenter BS and Cheek CH. 1970. Trace determination of uranium in biological material by fission track counting. Anal. Chem. 42:121-123.

Cathcart JB. 1956. Distribution and occurrence of uranium in the calcium phosphate zone of the landpebble phosphate district of Florida. Proceedings of the International Conference on Peaceful Uses of Atomic Energy, Geneva, 1955. 6:514-519.

Cathcart JB and Gulbrondsen RA. 1973. Phosphate deposit. U.S. mineral resources, U.S. Geological Survey Professional Paper 820, pp. 515-525.

Chen PS Jr., Terepka AR and Hodge HC. 1961. The pharmacology and toxicology of the bone seekers. Ann. Rev. Pharmacol. 1:369-396.

Cheney ES. 1981. The hunt for giant uranium deposits. Am. Sci., Jan.-Feb., pp. 37-48.

Cothern CR, Lappenbusch WL and Cotruvo JA. 1983. Health effects guidance for uranium in drinking water. Health Physics 44:377-384.

Cowan GA. 1976. A natural fission reactor. Sci. Am. 235:26-47.

CRPB. 1980. Canadian Radiation Protection Bureau. Guidelines for Canadian drinking water quality, 1978. Section on uranium. Supporting documentation. Health and Welfare, Ottawa, Canada.

de Rey BM, Lanfranchi HE and Cabrini RL. 1984. Deposition pattern and toxicity of subcutaneously implanted uranium dioxide in rats. Health Physics 46:688-692.

Donoghue JK, Dyson ED, Hislop JS, Leach AM and Spoor NL. 1972. Human exposure to natural radiation. Br. J. Ind. Med. 29:81-89.

Dounce AL. 1949. The mechanism of action of uranium compounds in the animal body. In: Voegtlin C and Hodge HC, eds. Pharmacology and toxicology of uranium compounds. National Nuclear Energy Series, Div. VI, Vol. 1, New York: McGraw-Hill. pp. 951-991.

Drury JS, Reynolds S, Owen PT, Ross RH and Ensminger JT. 1981. Uranium in U.S. surface, ground and domestic waters. EPA 570/9-81-001 by Health and Environmental Studies Program, Information Center Complex, Oak Ridge National Laboratory, Oak Ridge, Tennessee, under Interagency Agreement No. EPA 79-D-X0674, January 1981. Available from NTIS, U.S. Department of Commerce, Springfield, VA.

Durbin PW and Wrenn ME. 1975. Metabolism and effects of uranium in animals. In: Proceedings of the Conference on Occupational Health Experience with Uranium, U.S. Energy Research and Development Administration, ERDA 93/UC 41, pp. 65-129.

Eadie GG and Kaufman RF. 1977. Radiological evaluation of the effects of uranium mining and milling operations on selected ground water supplies in the grants mineral belt, New Mexico. Health Physics 32:231-241.

Edgington DN. 1967. The estimation of thorium and uranium at the submicrogram level in bone by neutron activation. International Journal of Applied Radiation and Isotopes 18:11-18.

Edgington DN. 1966. ANL 7060, Argonne National Laboratory, Review of July 1964 to June 1965.

Eve IS. 1964. Some suggested maximum permissible single intakes of uranium. Health Physics 10:773-776.

Filippova LG, Nifatov AP and Buldakov LA. 1980. Morphological changes in rats after administration of uranium-238 combined with plutonium-239 or americium-241. Radiobiologia 20:886-891.

Filippova LG, Nifatov AP and Lyubchanskiy ER. 1978. Radiobiologia 18:400-405.

Finkel MP. 1953. Relative biological effectiveness of radium and other alpha emitters in CF No. 1 female mice. Proceedings of the Society for Experimental Biology and Medicine 83:494-498.

Fisenne IM, Perry PM, Chu NY and Harley NH. 1983. Measured ^{234,238}U and fallout ^{239,240}Pu in human bone ash from Nepal and Australia: Skeletal alpha-dose. Health Physics 44 (Suppl. 1):457-467.

Fisenne IM, Perry PM and Welford GA. 1980. Determination of uranium isotopes in human bone ash. Analytical Chemistry 52:777-779.

Fish BR, Payne JA and Thompson JL. 1960. Ingestion of uranium compounds. In: Oak Ridge National Laboratory, Health Physics Division, Annual Report, Oak Ridge National Laboratory, Oak Ridge, TN. ORNL-2994. pp. 269-272.

Fix PF. 1956. Hydrogeochemical exploration for uranium. U.S. Geological Survey Professional Paper 33, pp. 667-671.

Fleischer RL. 1980. Isotopic disequilibrium of uranium; alpha-recoil damage and preferential solution effects. Science 207:979-981.

Fukuda S and Kopple JD. 1980. Chronic uremia syndrome in dogs induced with uranyl nitrate. Nephron. 25:139-143.

Ganguly AK. 1970. Assessment of internal radioactive contamination: The present trends n the Indian programs. The detection and assessment of uranium and plutonium in the whole body and in the critical organs. Vienna: International Atomic Energy Agency Report 118.

Ghadially FN, Lalonde JA and Yung-Steppuhn S. 1982. Uraniosomes produced in cultured rabbit kidney cells by uranyl acetate. Virchows Archiv. B. 39:21-30.

Giamond RJ. 1975. The radiological aspects of fertilizer utilization. Office of Radiation Programs, U.S. Environmental Protection Agency.

Goel KA, Garg VK and Garg V. 1980. Histopathology of kidney of albino rat poisoned with uranyl nitrate. Bulletin of Environmental Contamination and Toxicology 24:9-12.

Goel KA, Garg VK and Garg V. 1979. Histopathological alterations induced by uranyl nitrate in the liver of albino rat. Bulletin of Environmental Contamination and Toxicology 22:785-790.

Gus'kova VN, Bragina AN, Kupriyanova VM and Mashneva NI. 1966. A contribution to the substantiation of maximum permissible concentration of natural uranium in the water of open basins. Akademiva Meditsinskikh Nauk SSSR, Vestnik. No. 8, pp. 51-56.

Habashi F. 1970. Uranium phosphate rock. Publication 52. State of Montana Bureau of Mines and Geology.

Haley DP. 1982. Morphologic changes in uranyl nitrate-induced acute renal failure in saline- and water-drinking rats. Laboratory Investigation 46:196-208.

Haley DP, Bulger RE and Dobyan DC. 1982. The long-term effects of uranyl nitrate on the structure and function of the rat kidney. Virchows Archiv. B. 41:181-192.

Hamilton EI. 1972. The concentration of uranium in man and his diet. Health Physics 22:149-153.

Hamilton EI. 1971. The concentration and distribution of uranium in human skeletal tissues. Calc. Tiss. Res. 7:150-162.

Hamilton EI. 1970. Uranium content of normal blood. Nature 227:501-502.

Hamilton JG. 1948. The metabolic properties of the fission products and actinide elements. Review of Modern Physics 20:718-728.

Harrison JD and Stather JW. 1981. The gastrointestinal absorption of proactinium, uranium and neptunium in the hamster. Radiation Research 88:47-55.

Haseltine SD and Sileo L. 1983. Response of American black ducks to dietary uranium: A proposed substitute for lead shot. Journal of Wildlife Management 47:1124-1129.

Haven FL and Hodge HC. 1949. Toxicity following the parenteral administration of certain soluble uranium compounds. In: Voegtlin C and Hodge HC, eds. The pharmacology and toxicology of uranium compounds. National Nuclear Energy Series, Div. IV, Vol. I, New York: McGraw Hill. pp. 281-308.

Hodge HC. 1973. A history of uranium poisoning (1824-1942). In: Hodge HC, Stannard JN and Hursh JB, eds. Uranium, plutonium, transplutonic elements. New York: Springer-Verlag. pp. 5-68.

Hofman W, Steinhausler F and Pohl E. 1979. Dose calculations for the respiratory tract from inhaled natural radioactive nuclides as a function of age. Health Physics 37:517-532.

Holtzman RB, Urnezis PW, Padova A and Bobula CM, III. 1979. Contamination of the human food chain by uranium mill tailings piles. Nuclear Regulatory Commission, Washington, D.C. NUREF/CR-0758, ANL/ES-69.

Hursh JB, Neuman WF, Toribara T, Wilson H and Waterhouse C. 1969. Oral ingestion of uranium by man. Health Physics 17:619-621.

Hursh JB and Spoor NL. 1973. Data on man. In: Hodge HC, Stannard JN and Hursh JB, eds. Uranium, plutonium, transplutonic elements: Handbook of Experimental Pharmacology XXXVI. New York: Springer-Verlag. pp. 197-239.

ICRP 30. 1979. International Commission on Radiological Protection. Part I: Limits for intakes of radionuclides by workers. Publication 30. Oxford: Pergamon Press. Supplement Part 1, pp. 102-104.

ICRP 26. 1977. International Commission for Radiation Protection. Oxford: Pergamon Press.

ICRP 2. 1959. International Commission for Radiation Protection. Oxford: Pergamon Press.

Jadon A and Mathur R. 1983. Gametogenic count and histopathological effect of thorium nitrate and uranyl nitrate on mice testes. Andrologica 15:40-43.

Kahn B, Straub CP, Robbins PJ, Wellman HN, Seltzer RA and Telles NC. 1969. Retention of radiostrontium, strontium, calcium and phosphorous by infants. Pediatrics 43:651-756.

Katz JJ and Rabinowitch E. 1951. The chemistry of uranium the element, its binary and related compounds. New York: Dover Publications.

Kaufman MI, Rydell HS and Osmond JK. 1969. $^{234}U/^{238}U$ Disequilibrium as an aid to hydrologic study of the Floridan aquifer. J. Hydrol. 9:374-386.

Krieger HL and Whittaker EL. 1980. Prescribed procedure for measurement of radioactivity in drinking water. U.S. Environmental Protection Agency, EPA-600/4-80-032.

Kronfeld J. 1974. Uranium deposition and Th-234 alpha-recoil: an explanation for extreme U-234/U-238 fractionation within the Trinity aquifer. Earth Planet Sci. Lett. 21:327-330.

Labassi FP. 1979. Report of the interagency task force on ionity radiation. U.S. Department of Health, Education and Welfare.

Larsen RP, Bhattacharrya MH, Oldham RD, Morretti ES and Cohen N. 1984. Gastrointestinal absorption and retention of plutonium and uranium in the baboon. In: Argonne National Laboratory, Environmental Research Division, Annual Report, July 1982-June 1983, Argonne National Laboratory, Argonne, IL. ANL-83-100. pp. 51-60.

La Touche YD and Willis DL. 1984. Localization of uranium in the tissues of male Wistar rats following oral gavage with uranyl nitrate solutions. Health Physics 47:210, and private communication.

Lee SY, Hall SK and Bondietti EA. 1981. Methods of removing uranium from drinking water. II. Present municipal water treatment over potential removal methods. Oak Ridge National Laboratory, ORNL/EIS-194.

Leussenhop AJ, Gallimore JC, Sweet WH, Struxness EG and Robinson J. 1958. The toxicity in man of hexavalent uranium following intravenous administration. Am. J. Roentgenol. 79:83-100.

Lipsztein JL. 1981. An improved model for uranium metabolism in the primate. Ph.D. dissertation, New York University, New York, NY. 222 pp.

Longane JJ. 1966. Analytical chemistry of selected metallic elements. Holt Reinhold & Winston, New York.

Lopatkina AP. 1964. Characteristics of migration of uranium in the natural waters of humid regions and their use in the determination of the geochemical background for uranium. Geochem. Intl. 4-6:788-795.

Lucas HF, Jr. and Markun F. 1970. Thorium and uranium in blood, urine, and cigarettes. In: Argonne National Laboratory Annual Report, Radiological Sciences Division, Argonne National Laboratory, Argonne, IL. ANL-7760, Part 2. pp. 47-52.

Luckey TD, Venngopal B and Hutcheson D. 1975. Heavy metal toxicity, safety and hormology. Academic Prep, NY: George Thieme Publishers.

Malenchenko AF, Barkun NA and Guseva GF. 1978. Effect of uranium on the induction and course of experimental autoimmune orchitis and thyroiditis. Journal of Hygiene, Epidemiology, Microbiology and Immunology 22:268-277.

Marey AN, Yartseva EI and Moiseenko EI. 1966. Hyg. Sanit. (translation of Gig. Sanit.). 31:199-203.

Marshall JH, Groer PG and Schlenker RA. 1978. Dose to endosteal cells and relative distribution factors for radium-224 and plutonium-239 compared to radium-226. Health Physics 35:91-101.

Masuda K. 1971a. Intake and urinary excretion of uranium in non-occupationally exposed persons: I. Recovery of uranium in the daily diet and urine. Japanese Journal of Hygiene 26:434-437.

Masuda K. 1971b. Intake and urinary excretion of uranium in non-occupationally exposed persons: II. Uranium in the daily diet. Japanese Journal of Hygiene 26:438-441.

Masuda K. 1971c. Intake and urinary excretion of uranium in non-occupationally exposed persons: III. Urinary excretion of uranium. Japanese Journal of Hygiene 26:442-446.

Masuda K. 1971d. Intake and urinary excretion of uranium in non-occupationally exposed persons: IV. Discussions on dietary intake and urinary excretion of uranium. Japanese Journal of Hygiene 26:447-450.

Maynard EA, Downs WL and Hodge HC. 1953. Oral toxicity of uranium componds. In: Voegtlin C and Hodge HC, eds. Pharmacology and toxicology of uranium compounds. National Nuclear Energy Series, Div. VI, Vol. I, Part 3. New York: McGraw-Hill. pp. 1221-1369.

Mays CW, 1985, Cancer risks from the lifetime intake of Ra and U isotopes, Health Physics, May 1985.

Mendelsohn FAO and Smith EA. 1980. Intrarenal renin, angiotensin II, and plasma renin in rats with uranyl nitrate-induced and glycerol-induced acute renal failure. Kidney International 17:465-472.

Mentzel RG. 1986. Uranium, radium and thorium content in phosphate rocks and their possible radiation hazard. J. Agr. Food Chem. 16:231-234.

McKelvey VE and Corswell LD. 1965. Uranium in phosphoria formation. Proceedings International Conference on Peaceful Uses of Atomic Energy. Geneva. 1955. 6:551-561.

Miyake Y, Shrnhashi Y, Katsuragi Y, Konazawa T and Sujimura Y. 1964. Recent researchers in the fields of hydrosphere, atmosphere and nuclear geochemistry. Miyake Y and Koyana T, eds. Moryzen, Tokyo. pp. 127-141.

Moore WS. 1967. Amazon and Mississippi River concentrations of uranium, thorium and radium isotopes. Earth Planet Sci. Lett. 2:231.

Morrow P, Gelein R, Beiter H, Scott J, Picano J and Yuile C. 1982. Inhalation and intravenous studies of UF_6/UO_2F_2 in dogs. Health Physics 43:859-873.

NAS. 1980a. National Academy of Sciences. Drinking Water and Health. The effects on populations of exposure to low levels of ionizing radiation: 1980. Volume 5. Washington, D.C.: National Academy Press.

NAS. 1980b. National Academy of Sciences. National Academy of Sciences Report, Drinking Water and Health, Vol. 3. Toxicity of Selected Drinking Water Contamination, by the Toxicology Subcommittee of the Safe Drinking Water Committee.

NAS. 1977. National Academy of Sciences. Drinking Water and Health Recommendations. Fed. Regist., Monday, July 11, 1977, 132:35746.

Nachay et. al. 1979.

Nizet A. 1981. Influence of uranyl nitrate upon tubular reabsorption and glomerular filtration in blood perfused isolated dog kidneys. Pfluegers Arch. (European Journal of Physiology) 391:296-300.

Nomiyama K and Foulkes EC. 1968. Some effects of uranyl acetate on proximal tubular function in rabbit kidney. Toxicol. Appl. Pharmacol. 13:89-98.

Novikov YV. 1972. Hygienic standardization of the permissible amount of uranium in drinking water. Gig. Sanit. 37:12-17.

Novikov YV. 1970. On the hygenic standards for natural uranium in water. Hyg. Sanit. (translation of Gig. Sanit.) 37:13-17.

Novikov YV. 1967. The effect of low concentration of uranium in drinking water on the health of the population. Hyg. Sanit. (translation of Gig. Sanit.) 33:340-344.

Novikov YV and Abramova LN. 1969. Investigation of uranium content in various human tissues. Hyg. Sanit. 34:370-374. (in Russian)

Novikov YV, Pushkina NN and Tambovtseva AM. 1968. The effects on man of low concentrations of uranium. Hyg. Sanit. (translation of Gig. Sanit.) 33:340-344.

Novikov YV and Rezanov II. 1962. The content of uranium in drinking water of soil origin and its excretion in urine and feces. Hyg. Sanit. 27:103-105. (in Russian)

Novikov YV and Rezanov II. 1962. To the question of uranium content in drinking water from ground water and its discharge with urine and excrement. Gig. Sanit. 10:103-105.

Novikov YV and Yudina TV. 1970c. Data on the biological effect of small amounts of natural uranium in water. Hyg. Sanit. (translation of Gig. Sanit.) 35:63-71.

Nozaki T, Ichikowa M, Sasuga T and Inarida M. 1970b. Neutron activation analysis of uranium in human bone, drinking water and daily diet. J. Radioanalyt. Chem. 6:33-40.

Osburn WS. 1975. Primardial radionuclides: their distribution, movement and possible effect within terrestrial ecosystems. Health Physics 11:1275-1285.

Osmond JK, Kaufman MI and Cowart JB. 1974. Mixing volume calculations, sources and aging trends of Floridan aquifer water by uranium isotopic methods. Geochim. Cosmochim. Acta. 38:1083-1100.

Osmond JK, Rydell HS and Kaufman MI. 1969. Uranium disequilibrium in groundwater: an isotope dilution approach in hydrologic investigation. Science :997- .

Pelayo JC, Andrews PM, Coffey AK, Calcagno PL, Eisner GM and Jose PA. 1983. The influence of age on acute renal toxicity of uranyl nitrate in the dog. Pediatric Research 17:985-992.

PHS. 1965. Public Health Service. Task Force, Public Health Service Drinking Water Standards in Toxicology. Meeting October 19, 1965, Washington, D.C. Draft for uranium prepared by H.E. Stokinger.

Picer M and Strohal P. 1968. Determination of thorium and uranium in biological materials. Anal. Chim. Acta 40:131-136.

Roessler CE, Smith ZA, Bolch WF and Prince RJ. 1979. Uranium and radium^{226} in Florida phosphate materials. Health Physics 37:269-277.

Rona E, Gilpatrick LO and Jeffery LM. 1956. Uranium determination in sea water. Trans. Am. Geophys. U. 37:697-701.

Rona E and Urry WD. 1952. Radioactivity of ocean sediments. VIII. Radium and uranium contents of ocean and river waters. Am. J. Sci. 250:241-262.

Rowland RE, Stehney AF and Lucas HF. 1978. Dose response relationship for female radium dial workers. Radiation Research 76:368-383.

Sackett WM and Cook G. 1969. Uranium geochemistry of the Gulf of Mexico. Trans. Gulf Coast Assoc. Geol. Soc. 19:23.

Sackett WM, Mo T, Spalding RF and Exner ME. 1973. A reevaluation of the marine geochemistry of uranium in radioactive contamination of the marine environment. IAEA-SM-158/51.

Savannah River. 1979. Savannah River Semi-Annual Report, Hydrogeochemical and Stream Sediment Reconnaissance, Savannah River Laboratory, Aiken, SC, DPST-79-138-1.

Schlenker RA and Oltman BG. 1973. Fission track autoradiographs. In: Argonne National Laboratory Annual Report, Argonne National Laboratory, Argonne, IL. ANL-8060, Part II, pp. 163-168.

Sikov MR and Mahlum DD. 1968. Cross-placental transfer of selected actinides in the rat. Health Physics 14:205-208.

Singh NP, Lewis LL and Wrenn ME. 1983. Concentrations of uranium isotopes in human tissues from two states in the United States. In: Broerse JJ et al., eds. Proceedings of the 7th International Congress of Radiation Research. Amsterdam: Martinus Nijhoff. Paper E5-13.

Somayajulu BLK, Meghani CA and Borole DV. 1980. Uranium in drinking waters: rejection by human body. In: Patel, B, ed. Management of the environment. New Delhi: Wiley Eastern. pp. 528-532.

Spalding RF and Sackett WM. 1972. Uranium in runoff from the Gulf of Mexico distribution province anomalous concentrations. Science 175:629-631.

Spier FW and Whitwell JR. 1976. Dosimetry of ^{239} radium in man and animals, the health effects of plutonium and radium. Jer SS, ed. Salt Lake City, Utah: University of Utah, The J.W. Press, pp. 537-552.

Stefanov ZH and Yorkova TS. 1964. Some indices of acute uranium poisoning. Hyg. Sanit. (translation of Gig. Sanit.) 29:141-145.

Stevens WF, Bruienger W, Atherton DR, Smith JM and Taylor GN. 1980. The distribution and retention of hexavalent ^{233}U in the beagle. Radiation Research 83:109-126.

Sullivan MF. 1983. Gut-related studies of radionuclide toxicity. In: Pacific Northwest Laboratory Annual Report for 1982. Pacific Northwest Laboratory, Richland WA. PHL-4600, Pt. 1. pp. 95-99.

Sullivan MF. 1980a. Absorption of actinide elements from the gastrointestinal tract of rats, guinea pigs, and dogs. Health Physics 38:159-171.

Sullivan MF. 1980b. Absorption of actinide elements from the gastrointestinal tract of neonatal animals. Health Physics 38:173-185.

Sullivan MF and Gorham LS. 1982. Further studies of the absorption of actinide elements from the gastrointestinal tract of neonatal animals. Health Physics 43:509-519.

Sullivan RE, Nelson NS, Ellett WH, Dunning DF Jr., Leggett RW, Yalcintos MG and Eckerman KF. 1981. Estimates of health risk from exposure to radioactive pollutants, draft report to U.S. EPA/Office of Radiation Programs, November, ORNL/TM-7745.

Sullivan MF, and Gorham LS. 1983. Can information on the gastrointestinal absorption of actinide elements by neonatal rats, guinea pigs, dogs and swine be extrapolated to man? Health Physics, 44:411-417.

Sullivan MF. 1984. Gut related studies of radionuclide toxicity. In: Pacific Northwest Laboratory Annual Report for 1983. PNL Richland, WA, PNL-5000 PT. 1. pp.61-64.

Szocs E, Zahajszky T, Juszko J and Balint P. 1979. Intrarenal haemodynamics in uranyl nitrate-induced acute renal failure. Acta Physilogica Academiae Scientiarum Hungaricae 54:51-68.

Taylor DM, Bligh PH and Duggan MH. 1962. The absorption of calcium, strontium, barium and radium from the gastrointestinal tract of the rat. Biochem. J. 83:25-29.

Till JE, Kaye SV and Tobalka Jr. 1976. The toxicity of uranium and plutonium to the developing embryo of fish. ORNL-5160 UC-41, July 1976.

Tracy BL, Quinn JM, Gilman AP, Villenueve DC, Secours VE, Valli DE and Yagminaf AP. 1983. The metabolism of ingested uranium in mammals. In: Broerse, JJ, et al. eds. Proceedings of the 7th International Congress of Radiation Research. Amsterdam: Martinus Nijhoff. Paper E5-18.

Turekian KK and Chan LH. 1971. The marine geochemistry of the uranium isotopes, Th^{230} and Ra^{231} from activation analysis in geochemistry and cosmochemistry. Brunfeld AO and Steinnes F, eds. Universitetsforlaget, Oslo-Bergen-Tromso. pp. 311-320.

Tyrakowski T. 1979a. Disturbances in electrolyte transport in UO_2^{++} ion intoxication -- Model studies on the preliminary stage of toxic acute renal failure. II. The effect of uranyl acetate on the kidney in vivo. Acta Med. Pol. 20:307-316.

Tyrakowski T. 1979b. Disturbances in electrolyte transport in UO_2^{++} ion intoxication -- Model studies on the preliminary stage of toxic acute renal failure. III. Disturbances in Na^+ and K^+ ion excretion and decrease in glomerular filtration rate following uranyl acetate intoxication. Acta Med. Pol. 20:317-329.

UNSCEAR. 1977. United Nations Scientific Committee on the Effects of Atomic Radiation. Report to the General Assembly, sources and effects of ionizing radiation. New York: United Nations.

Wrenn ME. 1977. Internal dose estimates. International Symposium on Areas of High Natural Radioactivity. Pocos de Caldas, Brazil, June 16-20, 1975. Edited by Academia Brasileria de Ciencias, Rio de Janiero, RJ.

Wrenn MW, Durbin PW, Howard B, Lipsztein J, Rundo J, Still ET and Willis DL May 1985. Metabolism of ingested uranium and radium. Health Physics.

Yamamoto T, Masuda K and Onishi N. 1968. Studies on environmental contamination by uranium. I. Environmental survey of uranium in Kamisaibara Village, Okayama Prefecture. Journal of Radiation Research (Japan) 9:92-99.

Yamamoto T, Yunoki E, Yamakawa M and Shimizu M. 1973. Studies on environmental contamination by uranium: 3. Effect of carbonate ion on uranium absorption and desorption from soils. J. Radiat. Res. 14:219-224.

Yuile CL. 1973. Animal experiments. In: Hodge HC, Stannard JN and Hursh JB, eds. Uranium, plutonium, transplutonic elements. New York: Springer-Verlag. pp. 165-196.

APPENDIX I

SUMMARY OF EARLIER ANIMAL STUDIES

Table I-1 Selected Data on the Toxicity of Uranium Compounds

Species	Compound	Dose	Route	Effect	Reference
Dog	UNO_3	1 g	p.o.	nil	Gmelin (1825)
Rabbit	UNO_3	4 g	p.o.	emesis	
	UNO_3	1.4 g	p.o.	fatal	
	UNO_3	0.18 g	i.v.	fatal	
Dog, rabbit	UAc^c	0.5-1 g	p.o.	fatal	LeConte (1854)
Man	UNO_3	1.8 mg?	p.o.	in diabetes	Bradford (1860)
Man	UNO_3	0.6-350 mg repeated	oral	loose bowels flatulence	Blake (1868)
Rabbit, cat, pup	UNO_3	3 mg-2g (dilution?)	oral	fatal, glycosuria in 1 cat	
Dog	UA_c	0.25-2 g	p.o.	fatal	Rabuteau (1873)
Man	UNO_3	20 mg	p.o., t.i.d.	nil?	Carey (1874)
Rabbit, dog	UNO_3	3.5-4400 mg	subc	fatal	Chittenden (1888)
Dog	UNO_3	50-450 mg repeated	p.o.	LD	Chittanden and Lambert (1889)

Table I-1 - continued

Species	Compound	Dose	Route	Effect	Reference
Dog, cat, rabbit, goat, goose, dove, crow, chicken, frog	UNO_3 NaU tartrate[a] rabbit 0.83	U mg/kg dog 1.66 goat 1.66 cat 0.41 rat 0.41	p.o.	MLD	Woroschilsky (1889)
Man	UNO_3	?	p.o.	dyspepsia, loose bowels	West (1895)
	UNO_3	0.3g? (dilution?)	p.o., t.i.d.	nil	West (1896)
Man	UNO_3	0.7 g?	p.o.	diarrhea	Burton (1896) (dilution?) t.i.d. $UNO_3$0.5g?p.o., nil (dilution?) t.i.d.
Man	UNO_3	1.8 g?	p.o.	nil	Bond (1898) (dilution?)
Dog	UNO_3	120 mg	subc	glycosuria	Lepine (1903)
Rabbit	UNO_3	7.5 mg	subc	LD	Richter (1904, 1905)

Table I-1 - continued

Species	Compound	Dose	Route	Effect	Reference
Rabbit	UNO_3	0.5-7.5 mg	subc	LD	Blanck (1906)
Rabbit	UNO_3	5-500 mg 5	p.o. subc	nephritis nephritis	Fleckseder 1906, 1907)
Rabbit	UNO_3	5 mg	subc	LD	Georgopulos (1906)
Dog, cat	UNO_3' UA_c' NaU tartrate[a]	0.5-2 mg/kg	subc	LD	Kobert (1906)
Rabbit	UNO_3	7.5-10 mg repeated	subc	LD	Heineke and Meyerstein (1907)
Dog	UNO_3	10-15 mg, twice	subc	nephritis	Slegel (1907)
Rabbit	UNO_3	1-5 mg, single or repeated	--	nephritis	Christian (1908)
Rabbit	UNO_3	5 mg repeated	p.o., subc	edema	Shirokauer (1908)
Rabbit	UNO_3	1-5 mg repeated	subc	nephritis	Smith (1908)
Rabbit	UNO_3	6.3 to 39 mg/kg	--	nephritis	Takayasu (1908)
Rabbit	UNO_3	5 mg daily	--	nephritis	Bence (1909)

Table I-1 - continued

Species	Compound	Dose	Route	Effect	Reference
Guinea pig	UNO_3	0.25-5 mg, single or repeated	subc	nephritis	Dickson (1909)
Dog, rabbit	UNO_3	1.8-37.5 mg	subc	nephritis	Pearce (1909)
Guinea pig	UNO_3	0.5-15 mg, repeated	subc	some survival	Dickson (1910)
Dog	UNO_3, UAc	5-20 mg	subc	nephritis	Theohari and Giurea (1910)
Rabbit	UNO_3	2.5-20 mg	--	nephritis	
Rabbit	UNO_3	5 mg/kg max.	subc	often survival	Christian et al. (1911)
Guinea pig	UNO_3	0.5 mg/kg	subc	usually fatal	
Rabbit	UNO_3	2 mg/kg	subc	MLD	Boycott and Ryffle (1913)
Rabbit	UNO_3	1 mg/kg	subc	never LD	
Dogfish (*Mustelis canis*)	UNO_3	100 mg/kg 150 mg/kg	subc	5 day dead in 1-3 days	Denis (1913/1914)
Rabbit	UNO_3	0.35 mg	subc	delayed LD	Dunner and Siegfried (1920)

Table I-1 - continued

Species	Compound	Dose	Route	Effect	Reference
Rabbit	UNO_3	1 mg	subc	LD	Gil Y Gil (1923-1924)
Man	U salts	industrial exposure	inhalation, skin	anemia	DeLaet and Meurice (1925)
Dog	UNO_3	2 mg/kg	subc	LD	Garnier et al. (1928)
Rabbit	UNO_3	3 mg/kg or 10 mg/kg	subc	3/4 died	
Rabbit	UNO_3	1-4 mg/kg	subc	4/13-4/5 dead	Garnier and Marek (1929)
Rabbit	UNO_3	1-5 mg repeated	subc	nephritis	Smith (1908)
Rabbit	UNO_3	6.3 to 39 mg/kg	--	nephritis	Takayasu (1908)
Rabbit	UNO_3	5 mg daily	--	nephritis	Bence (1909)
Guinea pig	UNO_3	0.25-5 mg, single or repeated	subc	nephritis	Dickson (1909)
Rabbit	UNO_3, USO_4[b]	0.5-1.3 mg	injection	renal death	Messini (1929)

TABLE I-1 - continued

Species	Compound	Dose	Route	Effect	Reference
Rabbit	UNO_3	0.5 mg/kg 1 mg/kg 2-10 mg/kg 10 mg/kg	subc subc subc subc	male female 1/2 1/1 dead 12/17 6/29 dead 4/5 6/7 dead to all all dead	Garnier and Marek (1932)
Rabbit	UAc	0.05 mg U	intra-cisternal?	MLD	Pentschew and Kassowitz (1932)
Rabbit	UNO_3	1-4 mg/kg	subc	4/13-4/5 dead	Garnier and Marek (1929)
Rabbit	UNO_3, USO_4[b]	0.5-1.3 mg	injection	renal death	Messini (1929)
Rabbit	UNO_3	0.5 mg/kg 1 mg/kg 2-10 mg/kg 10 mg/kg	subc subc subc subc	male female 1/2 1/1 dead 12/17 6/29 dead 4/5 to 6/7 dead all all dead	Garnier and Marek (1932)
Rabbit	UAc	0.05 mg U	intra-cisternal?	MLD	Pentschew and Kassowitz (1932)
Rabbit	UNO_3	1 mg 0.83 mg 0.5 mg 0.35 mg	subc subc subc subc	LD-Mikawa LD-Sollmann LD-Angermayer LD-Pohl	Sato (1933)
Rabbit	UNO_3	5-30 mg/kg	subc	min LD to max LD	Vetri (1933)

TABLE I-1 - continued

Species	Compound	Dose	Route	Effect	Reference
Dog, kitten mouse, cat, guinea pig	"Coll-uranium"	1-15 mg/kg/day	subc	LD	Maloney and Burton (1938)

Table I-1 - continued - Tubular Injury

Species	Compound	Dose (Subc)	Reference
Dog, cat, rabbit, rat, goat, goose, dove, crow, chicken, frog	UNO_3, tartrate	0.5 mg to 1 g	Woroschilsky (1889)
Rabbit	UNO_3	7.5 mg	Richter (1905)
Rabbit	UNO_3	1 to 5 mg	Smith (1908)
Dog	UNO_3	1.5 mg/kg	Pearce et al. (1910)
Rabbit	UNO_3?	8-12 mg/kg	Schlayer and Takayasu (1910)
Rabbit	UNO_3	2.5 to 20 mg	Gross (1911)
Dog, cat, rabbit	UNO_3	0.35 to 1.4 mg	Pohl (1911/1912)
Rabbit, guinea pig, rat, mouse	UNO_3	---	Suzuki (1912)
Rabbit	UNO_3	2 mg/kg	Boycotta and Ryffle (1913)
Rabbit	UNO_3	10 mg	Ghoreyeb (1913)
Dog	UNO_3	8.5 to 30 mg	Kraus (1913)
Dog	UNO_3	3-12 mg/kg	O'Hare (1913)

Table I-1 - continued - Tubular Injury

Species	Compound	Dose (Subc)	Reference
Dog	UNO_3	6.7 mg/kg	MacNider (1912-1913, 1914, 1915, 1919)
Rabbit, guinea pig, rat	UNO_3	5 mg	Oliver (1915)
Guinea pig	UNO_3	100 mg/kg	Suzuki (1926)
Rabbit	UNO_3	7 mg	Auriat (1927)
Rabbit	UNO_3	6 mg, intrap.	Binet and Marek (1933)
Rabbit	UNO_3	0.5 mg/kg	Mentzer (1934)

Table I-1 - continued - Glomerular Injury (Minimal to Severe)

Species	Compound	Dose (Subc)	Reference
Dog, rabbit	UNO_3	3.5 to 440 mg p.o. 2.5 to 200 mg	Chittenden and Lambert (1888, 1889)
Rabbit	UNO_3	7.5 to 10 mg	Heineke and Meyerstein (1907)
Rabbit	UNO_3	5 mg	Shirokauer (1908)
Rabbit	?	6 to 39 mg	Takayasu (1908)
Guinea pig	UNO_3	0.25-5 mg	Dickson (1909)
Rabbit	UNO_3	1 to 5 mg	Christian et al. al. (1911)
Rabbit	UNO_3	0.43 to 0.7 mg intra-renal artery	Baehr (1912, 1912-1913)
Cat	UNO_3	0.8 to 2 mg	Folin et al. (1912)
Rabbit	UNO_3	3 to 5 mg/kg	Christian and O'Hare (1913)
Rabbit	UNO_3	12.5 to 25 mg intrap.	Hess and Wiesel (1913)

Table I-1 - continued - Glomerular Injury (Minimal to Severe)

Species	Compound	Dose (Subc)	Reference
Dog	UNO_3	6.7 mg/kg	MacNider (1912, 1913, 1920
Rabbit	UNO_3	3-12 mg/kg	O'Hare (1913)
Rabbit	?	?	Fahr (1914)
Rabbit	UNO_3	3 to 9 mg	Fitz (1914)
Dog	UNO_3	100 mg	Mosenthal (1914)
Rabbit	UNO_3	25 to 50 mg, intrap.	Wiesel and Hess (1915)
Rabbit	UNO_3	0.35 0.013 to 0.042 mg intra-renal artery	Dunner and Siegfried (1920)
Rabbit	UNO_3	25 mg, intrap.	Roth and Bloss (1922)
Cat	UNO_3	8 to 15 mg	Kellaway et al. (1925)
Rabbit	UNO_3	3.5 to 5 mg	Suzuki (1926)
Rabbit	UNO_3	0.4 to 452 mg	Dominguez (1928)
Dog	UNO_3	15 mg/kg	Milhorat and Deuel (1928)

Table I-1 - continued

Species	Compound	Dose (Subc)	Reference
Toad	UNO_3	6 mg	Gerard and Cordier (1932)
Rabbit	UNO_3	0.5 to 303 mg	Hunter and Roberts (1932)
Rabbit	UNO_3	10 mg/kg	Garnier and Marek (1931b)
Rabbit	UNO_3	i.v. and intrarenal artery	Patrossi and Rogers (1931)
Dog	UNO_3	0.2 to 3.6 mg/kg 1.1 to 6.3 mg/kg	Sachs (1934)
Guinea pig	UNO_3	8 to 100 mg/kg	
Rabbit	UNO_3	2.5 to 10 mg/kg	

Table I-1 - continued - Selected Reports of Experimental Edema in the Rabbits with Uranium Nephritis

Treatment	Uranium dose	Effect	Investigator
Sodium chloride water, Beets vs. Oats	UNO$_3$, 5-7.5 mg, subc, S, R^a	edema, pleural and peritoneal fluid	Richter (1904, 1905)
Serum from uranium nephritic		edema in recipient	Heineke (1905)
Serum from uranium nephritic	UNO$_3$, 5-7.5 mg, subc	inconstant incidence of edema in recipient	Blanck (1906)
Nephrectomy then serum from U nephritic	UNO$_3$, 5 mg, subc	edema	Georgopulos (1906)
No extra water		grades of edema	
Nephrectomy	then, UNO$_3$, 5 mg, intrap.	edema	Fleckseder (1906, 1907)
Repeated chromate and sodium chloride then a) control or b) U nephritic	UNO$_3$, 7.5 mg, subc, S, R	edema, slightly more often in U nephritic	Heineke and Meyerstein (1907)
Sodium chloride effective only on water-rich diet	UNO$_3$, 5 mg, subc, S, R	edema	Christian et al. (1908, 1909, 1911)
Some given water p.o.	UNO$_3$, 7.5 mg, subc.	edema and fluid only when water given	Pearce (1909)

Table I-1 - continued

Treatment	Uranium dose	Effect	Investigator
Fasted then A. Thirsty + U	UNO_3, 5 mg, subc?, R	no edema, loss of body weight, rbc or , serum refractive index (RI) or	Bence (1909)
B. Nephrectomized + water p.o. + U		edema, less loss of body weight, rbc, RI little change	
C. Water p.o. + U		edema, weight loss at times only 4-5%, rbc , RI	
D. Nephrectomized + water excess + U		usually edema, body weight usually , rbc , RI In each, blood volume most near death	
Acute U nephritis		enormous edema	Theohari and Giurea (1910)
i.v. infusion of hypotonic sodium chloride 4 days after U	?	no edema, sometimes ascites, Hgb, blood sodium chloride	Schmidt and Schlayer (1911)
Acute U nephritis	UNO_3, 1-5 mg, subc, S, R	1/16 rabbits with fluid in serous cavities, no subcutaneous edema. No heart failure	Dickson (1912)

Table I-1 - continued

Treatment	Uranium dose	Effect	Investigator
Subcutaneous infusion of sodium chloride then U	UNO_3, 0.3-20 mg/kg, subc	peritoneal and pleural fluid	Dake (1926)

Adapted from Hursh (1973).

Table I-2

Treatment	Uranium dose	Effect	Investigator
After U, 0.8 g NaCl in 20 mg H_2O	UNO_3, 7 mg. subc	edema, whether Cl, SO_4 acetate, PO_4, citrate, etc. also given. Not same as tieing ureters or nephrectomy	Mauriac and Aubel (1926)
Rapid vs. slow U poisoning NaCl p.o.	UNO_3, 5 mg, subc, R	slow: Na/Cl in blood and urine, more Na and less H_2O in muscle after NaCl p.o. rapid: NaCl in muscle with normal Na/Cl, K in blood and muscle	Aubel et al. (1926)
U nephritis	UNO_3, injected	straited muscle shows histological changes secondary to edema and not a uranium effect	Auriat (1927a)
Sodium given to some	UNO_3 7 mg, subc, R	some without edema, some with slight edema, abdominal, lumbar	Auriat (1927b)
Acute U nephritis fed after 2nd day, given 0.8 g NaCl	UNO_3, 7 mg, subc, R	marked edema with serous effusion	
Subacute, 3 weeks	3 weekly doses	degenerative lesions	

Table I-2 - continued

Treatment	Uranium dose	Effect	Investigator
Subacute total 2 months	3 weekly doses, then daily doses	loss of body weight, no edema	
Chronic U nephritis up to 6 months	UNO_3, 7 mg, subc. R	in edema, fluids are imbibed into tissues, because of a mineral complex disequilibrium	Aubel and Mauriac (1927)
U nephritis	UNO_3, 10 mg/kg, subc	edema about hilus of liver and root of mesentery	Whitney (1928)
Subcutaneous 0.1 cm^3 of isotonic NaCl in ear	UNO_3, 3-10 mg/kg, subc	more rapid disappearance in a nephritic than in control. Extreme thirst	Schulmann and Marek (1928)
Physiological NaCl or H_2O, p.o.	UNO_3, 7 mg, subc	less urine with physiological saline, more exudate with water	Govaerts (1928)
U nephritis, moderate or severe	UNO_3, 10, 15 mg, subc	little difference in blood or in plasma volume in mild poisoning. When severe, in blood and plasma vol.	Litzner (1930)
U nephritis	UNO_3, subc	edema formation not accompanied by other changes (e.g., in serine to globulin ratio). Hypoproteinemia no greater than in "dry" nephritic	Traissac (1932)

Table I-2 - continued

Treatment	Uranium dose	Effect	Investigator
Guinea pig given repeated doses, water not forced	UNO_3, 0.5-1 mg, subc, S, R	marked ascites in some, no edema in others	Dickson (1910, 1912)
Dog	UNO_3, 3-10 mg, subc, S, R	edema	
Dog given serum from renal vein	UNO_3	sometimes edema	Tria (1910)
Guinea pig given NaCl and water	UNO_3, 0.5-5 mg, S, R	edema in some	Christian et al. (1911)
Dog, U nephritis	U	no edema, marked increase in blood	Plesch (1922)
Dog	UNO_3, 1-4 mg/kg subc, S, R	no edema in dog although fluid in rabbit peritoneally, pleurally and pericardially	Garnier et al. (1928)

[a] S = Single; R = Repeated.

Table I-3 - Selected Reports of Human Administration (Usually UNO_3)

Reference	Dose	Comment
Bradford (1860)	gr. 2 or 3[a]	less urine; after continued use, less sugar
Carey (1874)	gr. 1/3 to 1/6 t.i.d.	single doses not over gr. 1-1/2, daily doses not over gr. 8
West (1895, 1896)	gr. 5 to 15, t.i.d.	slow acting; dyspepsia in 1 at gr. 5, t.i.d.; loose bowels
Burton (1896)	gr. 9 to 12, t.i.d.	diarrhea at gr. 12, not at gr. 9
Angermeyer (1897)	gr. 5 to 10	toxic hazard
Soundby (1897)	---	not effective in diabetes
Tyson (1897)	---	not effective in diabetes; loose bowels
Duncan (1897)	gr. 20 t.i.d. gr. 3 to 11	improved health of diabetics sometimes continued for 3 months
Bond (1897, 1898)	gr. 30 t.i.d.	lessened glycosuria; no symptoms in diabetic mental patients; no improvement of mental symptoms
Wilson (1903a and b)	---	increased uric acid excretion
Tylecote (1904)	gr. 1 t.i.d. initially	weight gain in phthisis; constipation
Kobert (1906)	---	U contraindicated in diabetes; total dose of 16 g. (?) UNO_3 in 100 days dangerous

Table I-3 - continued

Reference	Dose	Comment
Wilcox (1917)	gr. 1/2 to 3	reduced thirst, polyuria, glycosuria in 46 of 54 diabetic patients; no untoward symptoms; treatment extended sometimes for months or years

[a] Third trituration. This dilution was frequently specified although apparently dilutions as great as the sixth attenuation or more were used on occasion. Hughes (1886) stated that only the first and second dilutions "have hitherto been used".

Adapted from Hursh (1973).

APPENDIX II

ASTM FLUOROMETRIC METHOD FOR DETERMINING THE CONCENTRATION OF URANIUM IN DRINKING WATER

APPENDIX II

Designation. D 2907 - 75

Standard Test Methods for

MICROQUANTITIES OF URANIUM IN WATER BY FLUOROMETRY[1]

This Standard is issued under the fixed designation D 2907; the number immediately following the designation indicates the year of original adoption or, in the case of revision, the year of last revision. A number in parentheses indicates the year of last reapproval.

1. Scope and Application

1.1 These methods are applicable to the determination of microquantities of uranium in water in the concentration range from 0.005 to 50 mg/litre.

1.2 The uranium fluorescence is quenched by many cations and some anions in the sample; it is enhanced by a few cations. If interfering ions are present, a direct fluorometric measurement is not suitable, and an extraction method must be used to provide accurate results. The methods and their concentration ranges are as follows:

	Sections	Concentration Range, mg/litre
Method A—Direct Fluorometric Method	6 to 14	0.005 to 2
Method B—Extraction Method	15 to 23	0.04 to 50

2. Applicable Documents

2.1 *ASTM Standards:*

D 1066 Sampling Steam[2]

D 1129 Definitions of Terms Relating to Water[2]

D 1192 Specification for Equipment for Sampling Water and Steam[2]

D 1193 Specification for Reagent Water[2]

D 3370 Practices for Sampling Water[2]

3. Definitions

3.1 For definitions of terms used in these methods refer to Definitions D 1129.

4. Reagents

4.1 *Purity of Reagent*—Reagent grade chemicals shall be used in all tests. Unless otherwise indicated, it is intended that all reagents shall conform to the specifications of the Committee on Analytical Reagents of the American Chemical Society, where such specifications are available.[3] Other grades may be used, provided it is first ascertained that the reagent is of sufficiently high purity to permit its use without lessening the accuracy of the determination.

4.2 *Purity of Water*—Unless otherwise indicated, references to water shall be understood to mean reagent water conforming to Specification D 1193, Type III.

5. Sampling

5.1 Collect the samples in accordance with the applicable method of the American Society for Testing and Materials, as follows: Methods D 1066, D 1192, and D 3370.

5.2 To ensure continued solubility of the sample constituents, adjust the pH of the sample to approximately 2 with nitric acid (sp gr 1.42).

METHOD A—DIRECT FLUOROMETRIC METHOD

6. Application

6.1 This method is applicable to the determination of uranium in waters containing insufficient quantities of interfering ions to either enhance or quench the fluorescence of a fused uranium-fluoride disk. The range of the method is from 0.005 to 2.0 mg/litre. Although higher concentrations of uranium can be determined by this method, better precision and accuracy can be obtained with other procedures.[4]

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[1] These methods are under the jurisdiction of ASTM Committee D-19 on Water and are under the direct responsibility of Subcommittee D 19.04 on Methods of Radiochemical Analysis.

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[2] *Annual Book of ASTM Standards*, Part 31.

[3] "Reagent Chemicals, American Chemical Society Specifications," Am. Chemical Soc., Washington, D. C. For suggestions on the testing of reagents not listed by the American Chemical Society, see "Reagent Chemicals and Standards," by Joseph Rosin, D. Van Nostrand Co., Inc., New York, N. Y., and the "United States Pharmacopeia."

7. Summary of Method

7.1 The method is based on the measurement of the fluorescence of a fused disk of sodium fluoride, lithium fluoride, and uranium compound exposed to ultraviolet light. The intensity of the fluorescence is proportional to the uranium concentration.

7.2 An aliquot of the sample is pipeted into a platinum disk containing a sodium fluoride-lithium fluoride flux and evaporated to dryness. The mixture of sample and flux is fused with a blast burner, a muffle furnace, a tube furnace, or an induction heater. The fused disk is excited with an ultraviolet source over the wavelength range from 320 to 370 nm and the intensity of the fluorescence at 530 to 570 nm is measured by the fluorometer.

8. Interferences

8.1 There are many ions that interfere with this method. Small quantities of cadmium, chromium, cobalt, copper, iron, magnesium, manganese, nickel, lead, platinum, silicon, thorium, and zinc interfere by quenching the uranium fluorescence. Niobium and tantalum are reported to enhance the uranium fluorescence. In such cases use Method B.

9. Apparatus

9.1 *Blast Burner, Muffle Furnace, Tube Furnace, or Induction Heater*, capable of a 900°C temperature.

9.2 *Fluorometer*, having an excitation wavelength range from 320 to 370 nm and measuring the emission at a wavelength of 530 to 570 nm and capable of detecting 0.5 ng, or less, of uranium.

9.3 *Glasses*, didymium.

9.4 *Pellet Dispenser*, made by cutting a 1-ml hypodermic syringe so as to leave the full bore open.

9.5 *Pipet*—A 5-ml hypodermic syringe connected by flexible plastic tubing to a 0.5-ml Mohr pipet (graduated in 0.01-ml subdivisions) mounted on a ring stand.

9.6 *Platinum Disks*, the size and shape to be determined by the requirements of the fluorometer.

9.7 *Pyrometer*, with a suitable range for determining the fusion temperature of the flux.

10. Reagents

10.1 *Flux Mixture*—Mix 98 parts of sodium fluoride (NaF) and 2 parts of lithium fluoride (LiF) by weight until homogeneous. Several lots of NaF and LiF from different manufacturers should be tested to obtain material with a low blank reading and a high uranium sensitivity. Sufficient reagent to last several years should be obtained from the best lot. The powder should be sealed tightly to exclude moisture, during use and storage.

10.2 *Nitric Acid (1+1)*—Mix 1 volume of nitric acid HNO_3 (sp gr 1.42) with 1 volume of water.

10.3 *Nitric Acid (1+9)*—Mix 1 volume of HNO_3 (sp gr 1.42) with 9 volumes of water.

10.4 *Nitric Acid (1+99)*—Mix 1 volume of HNO_3 (sp gr 1.42) with 99 volumes of water.

10.5 *Potassium Pyrosulfate* ($K_2S_2O_7$), solid.

10.6 *Uranium Stock Solution (1 ml = 1 mg U)*—Dissolve 0.5896 g of uranous-uranic oxide (U_3O_8) in 20 ml of HNO_3 (1+1) and slowly evaporate to near dryness. Dissolve residue with 10 ml of HNO_3 (1+9) and quantitatively transfer to a 500-ml volumetric flask. Dilute to 500 ml with HNO_3 (1+99). Mix solution and transfer to a *clean* dry polyethylene bottle. This solution will contain 1000 mg U/litre.

10.7 *Uranium Stock Solution (1 ml = 0.05 mg U)*—Pipet 25 ml of the uranium solution (1 ml = 1 mg) into a 500-ml volumetric flask. Dilute to 500 ml with HNO_3 (1+99). Mix well and transfer to a *clean*, dry polyethylene bottle. This solution will contain 50 mg U/litre.

10.8 *Uranium Stock Solutions*—Prepare 200 ml each of standard solutions containing 10, 1, 0.5, 0.1, 0.05, and 0.005 mg/litre of uranium by diluting appropriate volumes of the uranium solution (1 ml = 0.05 mg U) with HNO_3 (1+99). Consecutive tenfold dilutions of the 10 and 5 mg/litre U standards are suggested for improved accuracy in preparing the more dilute standards. Mix each standard well and transfer to a *clean*, dry polyethylene container.

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[4] ASTM Method E 217, Uranium by Controlled Potential Coulometry, and ASTM Method E 318, Colorimetric Determination of Uranium in Aqueous Solutions, *Annual Book of ASTM Standards*, Part 45.

11. Calibration and Standardization

11.1 *Standardization of Fusion Operations*—The fusion operation is the most critical step in the fluorometric procedure. Small variations in the duration of the fusion, temperature of the fusion, and in the method of cooling the fused disk can cause large variations in the fluorescence. Therefore, it is imperative to standardize each step of the fusion operation to obtain reproducible results.

11.1.1 Choose the method of attaining the fusion temperature from one of four acceptable methods. These methods are burner fusion (either single or multiple fusions), induction heater fusion, muffle furnace fusion, or tube furnace fusion. Although reproducible results can be obtained with each method, the methods are not necessarily interchangeable. Use the same method of attaining the fusion temperature for the samples, blanks, and standards. If a burner is used for the fusion operation, use a reducing flame since an oxidizing flame increases the reaction rate between the flux and the platinum disk. The dissolved platinum will subsequently quench the fluorescence of the fused disk. Avoid excessively high temperature fusions or prolonged fusions, since it increases the amount of dissolved platinum in the fused disk. Very short fusions, however, can result in poor distribution of the uranium.

NOTE 1: **Caution**—Perform all fusion operations in a hood to avoid breathing the NaF-LiF fumes. Wear didymium glasses during burner fusions for eye protection and for ease in observing the molten flux.

11.1.2 Since the quantity of flux, which affects several variables in the fusion operation, cannot be specified in the procedure due to nonconformity in the size of fluorometer dishes, use the following criteria to standardize the fusion operation:

11.1.2.1 Determine the melting point of the flux with a pyrometer.

11.1.2.2 Standardize the fusion temperature at 50°C above the melting point of the flux.

11.1.2.3 Determine the quantity of flux that will completely wet the depression of the platinum dish during the fusion.

11.1.2.4 Determine the amount of time required to completely melt the quantity of flux established in 11.1.2.3 at the temperature established in 11.1.2.2.

11.1.2.5 Standardize the fusion time by multiplying the value obtained in 11.1.2.4 by 1.5 and rounding to the nearest 1 min.

11.1.2.6 Provide for an annealing or a slow cooling step following the fusion to give higher sensitivity and better reproducibility of the physical properties of the fused disk.

11.2 *Calibration of Fluorometer*—The instructions for the operation and adjustment of the fluorometer will be provided by the manufacturers. Although a daily factor is used to convert the fluorometer readings to mg U/litre for each method, perform a preliminary calibration to confirm a linear relationship between the fluorometer readings and the uranium concentrations.

11.2.1 Add a NaF-LiF flux pellet, using the quantity of flux established in 11.1.2.3, to 20 platinum dishes. These platinum dishes will be used for two blanks and nine duplicate uranium standards. Duplicate 0.1 and 0.2-ml aliquots of the 0.005, 0.05, 0.50, and 5.0 mg U/litre standards and duplicate 0.1 aliquots of the 50 mg U/litre solution shall be used to calibrate the fluorometer. These aliquots will simulate samples containing 0.005, 0.010, 0.050, 0.10, 0.50, 1.0, 5.0, 10.0, and 50.0 mg U/litre, respectively. After evaporation of the blanks and standards under an infrared lamp, fuse the blank and standard pellets in accordance with the standardized conditions established in 11.1.2. Cool the fused disks to room temperature, and either measure the fluorescence of the blanks and the standards with the fluorometer or zero the fluorometer with the blanks and measure the fluorescence of the standards. Follow the manufacturer's recommendations to either zero the fluorometer with the blank or record the reading of the blank. If the blanks have not been used to zero the fluorometer, subtract the average value of the blanks from the average readings of each standard. Plot the uranium concentration in mg/litre versus the product of the fluorometer readings and the scale factor for each standard. A straight line should be obtained.

11.2.2 It is necessary to recalibrate the fluorometer whenever any change is made in the fluorometer, reagents, or fusion conditions.

12. Procedure

12.1 Using the quantity of the NaF-LiF flux established in 11.1.2.3, add a flux pellet to each of the eight platinum dishes with the pellet dispenser. These platinum dishes will be used for two blanks and six uranium standards. Add a pellet of the flux to each platinum dish required for the samples using two platinum dishes per sample.

12.2 Pipet duplicate 0.1-ml aliquots of the 0.01, 0.10, and 1.0 mg/litre uranium standards into six of the platinum dishes.

12.3 Pipet duplicate 0.1-ml aliquots of the samples into platinum dishes.

12.4 Evaporate samples and standards to dryness under an infrared lamp.

12.5 Using the fusion operation established in 12.1.2, fuse the samples, blanks, and uranium standards.

12.6 After the fused disks have cooled to room temperature, measure the fluorescence of the samples, blanks, and uranium standards with the fluorometer.

12.7 *Cleaning the Platinum Dishes*—Remove the disk from the platinum dish and wash the dish in hot water. Fuse each dish with potassium pyrosulfate ($K_2S_2O_7$), cool, and dissolve the residue in hot water. Store the dishes in dilute HNO_3 (1+9) until needed. Rinse in water prior to use.

13. Calculation

13.1 Calculate the uranium concentration in milligrams per litre as follows:

$$\text{Uranium, mg/litre} = F \times R \times S$$

where

R = fluorometer reading of the sample,
S = fluorometer scale, and
F = $[0.01/(R_1 \times S_1)] + [0.10/(R_2 \times S_2)] + [1.0/(R_3 \times S_3)]/3$

where

0.01, 0.10, and 1.0 = mg/litre of the three uranium standards,
R_1, R_2, and R_3 = fluorometer reading of the three standards, and
S_1, S_2, and S_3 = fluorometer scale used for the three uranium standards.

NOTE 2—If the fluorometer is not zeroed with the blank, subtract the reading of the blank from the reading of the samples and the uranium standards.

14. Precision[4]

14.1 The precision of the method varies with the quantity of uranium being determined as follows:

$$s_o = 0.1144\, x^{1.2468} + 0.0016$$

or

$$(\log s_o - 0.0016) = \log 0.114 + 1.2468 \log x$$

and

$$s_t = 0.2001\, x^{1.5293} + 0.0024$$

or

$$(\log s_t - 0.0024) = \log 0.2001 + 1.5293 \log x$$

where

s_o = single-operator precision, mg/litre,
s_t = overall precision, mg/litre, and
x = uranium concentration, mg/litre

METHOD B—EXTRACTION METHOD

15. Application

15.1 This method is applicable to the determination of uranium in waters known to contain sufficient quantities of impurities to interfere with a direct fluorometric determination. The range of the method is from 0.05 to 50 mg/litre. Although this range can be extended, better precision and accuracy can be obtained at 50 mg/litre and above with other procedures.[4]

16. Summary of Method

16.1 The uranium in the sample is separated by an extraction with hexone (methyl isobutyl ketone) using an acid-deficient aluminum nitrate salting solution containing tetrapropylammonium nitrate (TPAN). The extracted uranium is fused with a sodium fluoride-lithium fluoride pellet. The uranium content is determined by measuring the ultraviolet activated fluorescence of the fused disk with a fluorometer. The fluorometer is calibrated with standard uranium solutions that have been extracted by the same procedure.

17. Interferences

17.1 Excessive quantities of sulfate ion or hydrogen ion interfere with this method by inhibiting the extraction of the uranium. These interferences are eliminated by evaporating the acidified solution to near dryness and dissolving the residue in nitric acid (38+62). With a specified sample aliquot, the tolerance limits are 5 *M* for sulfate and 16 *N* for acid. Alkaline solutions should be acidified with concentrated nitric acid before processing with the analysis.

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[4] Supporting data for these methods have been filed at ASTM Headquarters as RR: D 19-1005.

18. Apparatus

18.1 For a description of the furnace, pellet dispenser, platinum dishes, pyrometer, and accessories required for the preparation of fused disks, see Section 9.

18.2 *Fluorometer*—See 9.2.

18.3 *Mechanical Test Tube Shaker.*

18.4 *Pipet*—See 9.5.

19. Reagents

19.1 *Acetone.*

19.2 *Aluminum Nitrate* [$Al(NO_3)_3 \cdot 9H_2O$], crystalline.

19.3 *Ammonium Hydroxide (sp gr 0.90)*—Concentrated ammonium hydroxide (NH_4OH).

19.4 *Flux Mixture*—See 10.1.

19.5 *Hexone (methyl isobutyl ketone).*

19.6 *Hydroxylamine Hydrochloride Solution (139 g/litre)*—Dissolve 13.9 g of hydroxylamine hydrochloride ($NH_2OH \cdot HCl$) in water and dilute to 100 ml.

19.7 *Nitric Acid (sp gr 1.42)*—Concentrated nitric acid (HNO_3).

19.8 *Nitric Acid (38+62)*—Mix 380 ml of HNO_3 (sp gr 1.42) with 620 ml of water.

19.9 *Potassium Permanganate Solution (31.6 g/litre)*—Dissolve 7.9 g of potassium permanganate ($KMnO_4$) in water and dilute to 250 ml.

19.10 *Tetrapropylammonium Hydroxide* (TPAH).

19.11 *TPAH Salting Solution*—Dissolve 1050 g of aluminum nitrate [$Al(NO_3)_3 \cdot 9H_2O$] in 700 ml of water with the aid of heat. Add 135 ml of NH_4OH (sp gr 0.90) and 10 ml of a 10 % solution of tetrapropylammonium hydroxide and dilute to about 950 ml with water. Stir until dissolved, then extract with 250 ml of hexone in a separatory funnel and discard extract. Filter the aqueous phase through a large fine porosity, sintered-glass Büchner funnel, add 10 ml of 10 % tetrapropylammonium hydroxide, and dilute to 1 litre with water.

19.12 *Uranium Standards*—See 10.7 and 10.8.

20. Calibration and Standardization

20.1 *Standardization of Fusion Operation*—Follow the procedure given in 11.1.

20.2 *Calibration of Fluorometer*—Proceed as directed in 11.2 except that the 0.005-ml/litre uranium standard may be omitted.

21. Procedure

21.1 If the sulfate concentration exceeds 5 *M* or if the acid concentration exceeds 16 *N*, evaporate an 0.5-ml aliquot of the sample to near dryness and redissolve in 0.15 ml of HNO_3 (38+62).

21.2 Pipet 0.5 ml of the sample, or transfer the redissolved aliquot from 21.1, into a 13 by 100-mm test tube. For neutral or alkaline samples, add 0.2 ml of HNO_3 (sp gr 1.42). With each group of samples, include two blanks and duplicate 0.5-ml aliquots of the 0.10, 1.0, and 10.0 mg/litre uranium standards.

21.3 While swirling, add 0.2 *N* $KMnO_4$ solution, 1 drop at a time, until the pink color persists.

21.4 While swirling, add 1 drop of $NH_2OH \cdot HCl$ solution. If the pink color persists, add another drop. If the pink color still persists, too much $KMnO_4$ solution was added in 21.3. Start again with a fresh aliquot.

21.5 Add 4.0 ml of the TPAH salting solution.

21.6 Pipet 2.0 ml of hexone into the test tube, stopper, mix well for 3 min with a mechanical mixer or 1 min if hand mixed. Allow to stand until the phases separate. If complete separation does not occur, centrifuge for 2 min.

21.7 Place a pellet of the fusion flux into each of two platinum dishes.

21.8 Pipet 0.2 ml of the organic phase onto each of the two pellets. Each time, rinse the pipet with acetone and add the rinsings to the pellet.

21.9 Continue as directed in 12.4 to 12.7.

22. Calculation

22.1 See Section 13. The 0.10-, 1.0 and 10.0 mg U/litre standards are used to calculate the factor *F*.

23. Precision

23.1 The precision of the method varies with the quantity of uranium being determined as follows:

$$s_o = 0.1691\,x^{0.9397}$$

or

$$\log s_o = \log 0.1691 + 0.9397 \log x$$

and

$$s_t = 0.2369\,x^{0.9551}$$

or

$$\log s_t = \log 0.2369 + 0.9551 \log x$$

where.

s_o = single-operator precision, mg/litre.

s_t = overall precision, mg/litre, and

x = uranium concentration, mg/litre

APPENDIX

X1. SUMMARY OF ROUND-ROBIN TESTING OF DIRECT FLUOROMETRIC URANIUM METHOD

N	Theory mg U/litre	Found mg U/litre	s_o mg U/litre	Predicted[a] s_o mg U/litre	s_t mg U/litre	Predicted[b] s_t mg U/litre
24	0.0063	0.0074	0.0019	0.0019	0.0026	0.0025
26	0.013	0.019	0.002	0.002	0.003	0.003
21	0.026	0.031	0.009	0.003	0.012	0.003
18	0.125	0.120	0.009	0.010	0.012	0.010
21	0.523	0.544	0.053	0.055	0.072	0.081
21	2.09	2.27	0.35	0.32	0.72	0.70

---

[a] Formula $s_o - 0.0016 = 0.1144\,x^{1.2468}$ or
$\log (s_o - 0.0016) = \log 0.1144 + 1.2468 \log x$
x = uranium concentration

[b] Formula $s_t - 0.0024 = 0.2001\,x^{1.5293}$ or
$\log (s_t - 0.0024) = \log 0.2001 + 1.5293 \log x$

X1.1 The 0.0016 and 0.0024 values for the formulas for calculating the s_o and s_t were determined by trial and error. The other constants were determined with a computer programmed for the least-squares fit for a curve of the general type, $y = ax^b$.

X1.2 A plot of the standard deviations versus uranium concentrations on log-log paper indicated that the s_o and s_t for the 0.026 mg U/litre standard were biased high. These two points were disregarded in the determination of the formulas for s_o and s_t.

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APPENDIX III

URANIUM ANALYTICAL METHODS FROM "PRESCRIBED PROCEDURES FOR MEASUREMENT OF RADIOACTIIVTY IN DRINKING WATER" EPA-600/4-800-032

APPENDIX III - RADIOCHEMICAL AND FLUOROMETRIC METHODS FOR URANIUM IN DRINKING WATER[a]

SECTION 12
URANIUM IN DRINKING WATER
RADIOCHEMICAL METHOD
METHOD 908.0

1. Scope and Application

1.1 This method covers the measurement of total uranium alpha particle activity in drinking water. Most drinking water sources, especially ground water sources, contain soluble carbonates and bicarbonates which complex and keep uranium in the water in solution.

1.2 Uranium isotopic abundances in drinking water sources are apt to be present in ratios in the deposits from which the uranium entered the water sources. The two predominant natural alpha emitting isotopes of uranium are uranium-234 and uranium-238. Uranium-238 is the predominant mass abundant isotope: greater than 99% compared to about 0.006% for uranium-234. However, uranium-234 has a specific activity for alpha particle emission that is 1.8 x 10^4 times greater than that of uranium-238. For an equilibrium condition, the activity of the uranium-234 is equal to that of the uranium-238. Therefore, the uranium mass concentration in water is not related to the alpha particle activity of the water.

1.3 The Drinking Water Regulations under the Safe Drinking Water Act, PL 93-523, require a measurement of uranium for drinking water samples that have a gross alpha activity greater than 15 pCi/L. A mass uranium concentration measurement of a water sample cannot be converted to uranium alpha activity without first analyzing for isotopic abundances. Therefore, a method such as this one is needed to determine the total uranium alpha activity of the sample, without doing an isotopic uranium analysis.

2. Summary of Method

2.1 The water sample is made acid by adding HCl and the sample is boiled to eliminate carbonate and bicarbonate ions. Uranium is coprecipitated with ferric hydroxide and separated from the sample. The uranium is then separated from other radionuclides which were carried down with the ferric hydroxide by dissolving the hydroxide precipitate in 8N HCl, putting the solution through an anion exchange column, washing the column with 8N HCl, and finally eluting the uranium with 0.1N HCl. The uranium eluate is evaporated and the uranium chemical form is converted to nitrate. The residue is transferred to a stainless steel planchet, dried, flamed, and counted for alpha particle activity.

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[a] From Krieger and Whittaker (1980).

2.2 Uranium recovery is determined with blank samples spiked with known amounts of uranium and taken through the procedure as a regular sample.

2.3 Counting efficiency is determined by transferring measured aliquots of an uranium standard to a planchet, diluting with 6-8 ml of a 1 mg/ml HIO_3 solution in 4N HNO_3, evaporating to dryness, flaming the planchet, and counting in an alpha counter.

3. Sample Handling and Preservation

3.1 Although carbonate ions in a water sample will help to keep uranium in solution, the addition of extra carbonate or bicarbonate ions to the sample as a preservative is not recommended because an increased carbonate concentration in the sample may cause some precipitation. Therefore, it is recommended that samples be preserved with HCl to pH 2 at the time of collection.

3.2 A sample size of at least 1 liter should be collected for uranium analysis.

4. Interferences

4.1 The only alpha-emitting radionuclide that may come through the chemistry and cause interference would be protactinium-231. However, protactinium-231 results from the decay of uranium-235, a low abundance natural isotope of uranium, and would therefore cause only a very small interference.

4.2 Since uranium is a naturally occurring radionuclide, reagents must be checked for uranium contamination by analyzing a complete reagent blank by the same procedure as used for the samples.

5. Apparatus - See Appendix D for details and specifications

5.1 Gas-flow proportional counting system or

5.2 Scintillation detection system.

5.3 Glassware.

5.4 Electric hot plate.

5.5 Ion exchange column: approximately 13 mm (i.d.) x 150 mm long with a 100 ml reservoir.

5.6 Stainless steel counting planchets, 2 inch diameter by 1/4 inch deep.

5.7 Millipore filter apparatus, 47 mm.

6. Reagents

6.1 All chemicals should be of reagent grade or equivalent whenever they are commercially available.

6.2 Ammonium hydroxide, 6$\underline{N}$: Mix 2 volumes 15$\underline{N}$ NH_4OH (conc.) with 3 volumes of water (carbonate-free).

6.3 Anion exchange resin - Strongly basic, styrene, quaternary ammonium salt, 4% crosslinked, 100-200 mesh, chloride form (such as Dowes 1x4, or equivalent).

6.4 Ferric chloride carrier, 20 mg Fe^{+3}/ml: Dissolve 9.6g of $FeCl_3.6H_2O$ in 100 ml of 0.5 $\underline{N}$ NCl.

6.5 Hydriodic acid: HI (conc.), sp. gr. 1.5, 47%.

6.6 Hydrochloric acid, 12$\underline{N}$: NCl (conc.), sp. gr. 1.19, 37.2%.

6.7 Hydrochloric acid, 8$\underline{N}$: Mix 2 volumes 12$\underline{N}$ HCl (conc.) with 1 volume of water.

6.8 Hydrochloric acid, 6$\underline{N}$: Mix 1 volume 12$\underline{N}$ HCl (conc.) with 1 volume of water.

6.9 Hydrochloric acid, 0.1$\underline{N}$: Mix 1 volume 0.5$\underline{N}$ HCl with 4 volumes of water.

6.10 Iodic acid, 1 mg/ml: Dissolve 100 mg HIO_3 in 100 ml 4$\underline{N}$ HNO_3.

6.11 Nitric acid, 16$\underline{N}$: HNO_3 (conc.), sp. gr. 1.42, 70.4%.

6.12 Nitric acid, 4N: Mix 1 volume 16N HNO_3 (conc.) with 3 volumes of water.

6.13 Sodium hydrogen sulfite, $NaHSO_3$.

6.14 Sodium hydrogen sulfite, 1% in HCl: Dissolve 1g $NaHSO_3$ in 100 ml 6N HCl.

7. Calibrations

7.1 Determine a counting efficiency (E), for a known amount of standard uranium (about 1000 dpm) evaporated from a 6-8 ml volume of a 1 mg/ml HIO_3 solution in a 2 inch diameter stainless steel planchet. If the standard solution is HCl solution, then aliquot portions of that solution must be converted to nitrate/HNO_3 solutions, eliminating all chloride ions from the solutions. This can be done by three successive evaporations after adding 5 ml portions of 16N HNO_3 to aliquot portions of the standard in small beakers (avoiding dry baking of the evaporated residue). The final solutions of the standard aliquots are made by adding 2 ml 4N HNO_3 solution to the third evaporated residues. Transfer the uranium standard a liquot solutions to 2 inch diameter stainless steel planchets.

Complete the transfer by rinsing the beakers two times with 2 ml portions of 4N HNO_3 and evaporate to dryness. Flame the planchets and count for at least 50 minutes for alpha particle activity. A reagent blank should be run along with the standard aliquots and should be alpha counted.

$$\text{Efficiency, cpm/dpm, (E)} = \frac{A - B}{C}$$

where:

A = gross cpm for standard
B = cpm for instrument background
C = dpm of standard used

7.2 A uranium recovery factor, R, is determined by the following procedure: Spike one liter tap water samples with aliquots of uranium standard solution (500-1000 dpm per sample). Take these spiked samples and a tap water blank through the entire procedure and count the separated and evaporated uranium for alpha particle activity.

$$\text{Recovery factor, (R)} = \frac{(F - B)}{CE}$$

where:
C = dpm of uranium standard added
F = gross cpm of spiked sample
B = cpm of reagent blank
E = efficiency factor, cpm/dpm

8. Procedure

8.1 Measure the volume of approximately one liter of the water sample to be analyzed.

8.1.2 If the sample has not been acidified, add 5 ml 12N HCl. and 1 ml ferric chloride carrier.

8.1.3 Mix the sample completely and use pH paper to check the hydrogen-ion concentration. If the pH is > 1, add 12N HCl until it reaches this value.

8.1.4 Cover with a watch glass and heat the water sample to boiling for 20 minutes.

8.1.5 The pH must be checked again after boiling and if it is > 1, 12N HCl must be added to bring the pH back to 1.

8.1.6 While the sample is still boiling gently add 6N NH_4OH to the sample from a polyethylene squeeze bottle with the bottle delivery tube inserted between the watch glass and the pouring lip of the beaker. The boiling action of the sample provides sufficient stirring action. Add 6N NH_4OH until turbidity persists while boiling continues; then add an additional 10 ml (estimated addition from the squeeze bottle).

8.1.7 Continue to boil the sample for 10 minutes more; then set it aside for 30 minutes to cool and settle.

8.1.8 After the sample has settled sufficiently, decant and filter the supernate through a 47 mm 0.45 micron membrane filter, using the larger millipore filtering apparatus.

8.1.9 Slurry the remaining precipitate, transfer to the filtering apparatus and filter with suction.

8.1.10 Place the filtering apparatus over a clean 250 ml filtering flask, add 25 ml 8N HCl to dissolve precipitate, and filter the solution.

8.1.11 Add another 25 ml 8N HCl to wash the filter, and then filter.

8.1.12 Transfer solution to the 100 ml reservoir of the ion exchange column.

8.1.13 Rinse the side arm filtering flask twice with 25 ml portions of 8N HCl. Combine in the ion exchange reservoir.

8.2 Anion Exchange Separation

8.2.1 Prepare the column by slurrying the anion exchange resin with 8N HCl and pouring it onto a column of about 13 mm inside diameter. The height of the resin bed should be about 80 mm.

8.2.2 Pass the sample solution through the anion exchange resin column at a flow rate not to exceed 5 ml/min.

8.2.3 After the sample has passed through the column, elute the iron (and plutonium if present) with 6 column volumes of 8N HCl containing 1 ml 47% HI per 9 ml of 8N HCL (freshly prepared).

8.2.4 Wash the column with an additional two column volumes of 8N HCl.

8.2.5 Elute the uranium with six column volumes of 0.1N HCl.

8.2.6 Evaporate the acid eluate to near dryness and convert the residue salts to nitrates by three successive treatments with 5 ml portions of 16N HNO_3, evaporating to near dryness each time.

8.2.7 Dissolve the residue (may be very little visible residue) in 2 ml 4N HNO_3.

8.2.8 Transfer the residue solution, using a Pasteur pipet, to a marked planchet, and complete the transfer by rinsing the sample beaker three times with 2 ml portions of 4N HNO_3.

8.2.9 Evaporate the contents in the planchet to dryness, flame to remove traces of HIO_3, cool, and count for alpha particle activity.

8.3 Column Regeneration

8.3.1 Pass three column volumes of 1% $NaHSO_3$ in 6N HCl through the column.

8.3.2 Pass six column volumes of 6N HCl through the column.

8.3.3 Pass three column volumes of water through the column.

8.3.4 Pass six column volumes of 8N HCl through the column to equilibrate and ready the resin for the next set of samples.

9. Calculations

$$\text{Uranium alpha activity, pCi/L} = \frac{(S - B) \times 1000}{2.22 \times E\ R\ V}$$

where:

S = gross cpm for sample
B = cpm of reagent blank
V = volume of sample used, ml
E = efficiency, cpm/dpm
R = recovery factor
2.22 = conversion factor for dpm/pCi

10. Precision and Accuracy

In a single laboratory test of this method, a stock uranium solution was prepared using tap water and spiked with an NBS uranium standard. The calculated concentration was 26.7 pCi/L. This stock solution was acidified with HCl as a preservative. Nine 1-liter aliquots were withdrawn and the procedure tested. Individual results were 22.4, 22.5, 24.0, 25.9, 2f 9, 26.5, 24.6, 25.7 and 23.9 pCi/L. The average concentration was 24.7 pCi/L with a standard deviation of 1.7 pCi/L. From these data, the method shows a negative 7.4% bias and a precision of ± 6.7% without the correction of the recovery factor.

References

Bishop CT et al. 1979. Radiometric method for the determination of uranium in water. EPA 600/7-79-093, EMSL-LV.

Edwards KW. 1968. Isotopic analysis of uranium in natural waters by alpha spectrometry. Radiochemical analysis of water. Geological Survey Water Supply Paper. 1696-F. Washington, D.C.: U.S. Government Printing Office.

SECTION 13
URANIUM IN DRINKING WATER - FLUORMETRIC METHOD
METHOD 908.1

1. Scope and Application

1.1 The method covers the determination of soluble uranium in waters at concentrations greater than 0.1 microgram/l. There is no upper limit, since waters whose uranium concentrations exceed the upper limit of the measurement range need only be diluted to be within this range.

1.2 Uranium is present in surface and ground waters at concentrations generally less than 20 microgram/l. This method is applicable for the monitoring of water discharges from industries related to the uranium fuel cycle. Since the method measures the mass of uranium, it is applicable to the assessment of chemical toxicity. The method can be indirectly used for the assessment of radiation effects if the isotopic composition is known or measured.

2. Summary of Method

Uranium is concentrated by coprecipitation with aluminum phosphate. The alumiunum phosphate is dissolved in dilute nitric acid containing magnesium nitrate as a salting agent and the coprecipitated uranium is extacted into ethyl acetate. After the ethyl acetate is removed by evaporation, the extracted residue is dissolved in nitric acid and diluted to volume in a small volumetric flask. Aliquots are transferred to each of two fusion dishes and dried. To one dish is added a known mass of uranium (0.1 micrograms) and dried. To one dish is added a known mass of uranium (0.1 micrograms) and dried. Flux containing sodium fluoride is added to each of the dishes, fused at a prescribed temperature, cooled and read in a fluorometer. The use of the standard addition technique corrects for any interference that may coextract with uranium.

3. Interferences

3.1 The fluorescence of uranium in a fluoride matrix can be quenched or enhanced by either cations or anions. When uranium is present in low concentration (less than 20 micrograms/l) these interferences can be eliminated by the coprecipitation of uranium on aluminum phosphate and subsequent uranium extraction into ethyl acetate.

3.2 Carbonate ions form soluble uranium complexes which prevent the coprecipitation on aluminum phosphate. Carbonates are removed by acidification and expelled from solution as volatile carbon dioxide.

4. Apparatus - See Appendix D for details and specifications

4.1 Fluorometer, Jarrel-Ash or equivalent.

4.2 Dish forming die, Cat. No. 26100, Fisher Scientific.

4.3 Fusion dish blanks - Gold or Platinum 0.015" thickness x 0.75" diameter.

4.4 Muffle furnace - controlled temperature.

4.5 Microliter pipette - 100 ul.

4.6 Glassware.

5. Reagents

5.1 Purity of Reagents - Reagent grade chemicals shall be used in all tests. Unless otherwise indicated, it is intended that all reagents shall conform to the specifications of the committee on analytical reagents of the American Chemical Society. Other grades may be used provided it is first ascertained that the reagent is of sufficiently high purity to permit its use without lessening the accuracy of the determination.

5.2 Purity of Water. Unless otherwise indicated, reference to water shall be understood to mean conforming to ASTM Specification D 1193, Type III.

5.3 Aluminum nitrate, 0.08M: Dissolve 15g $Al(NO_3)_3.9H_2O$ in 500 1 of water.

5.4 Ammonium hydroxide, 15N: NH_4OH (conc.) sp.gr. 0.90, 56.6%.

5.5 Diammonium hydrogen phosphate 0.11M: Dissolve 7.26g $(NH_4)_2HPO_4$ in 500 ml water.

5.6 Ethyl acetate, $CH_3COOC_2H_5$, reagent grade.

5.7 Flux: Mix together 9 parts NaF, 45.5 parts Na_2CO_3, 45.5 parts K_2CO_3 by weight in a ball mill.

5.8 Magnesium nitrate, 3.5M: Dissolve 449g $Mg(NO_3)_2.6H_2O$ in 350 ml water containing 32 ml 16N HNO. Warm if necessary to dissolve. Cool and dilute to 500 ml.

5.9 Nitric acid, 16N: HNO_3 (conc.), sp. gr. 1.42, 70.4%.

5.10 Nitric acid, 0.1N: Mix 1 volume 16N HNO_3 (conc.) with 159 volumes of water.

5.11 Phenolphthalein (5 g/l): Dissolve 0.5 g phenolphthalein in 50 ml ethanol (95%) and dilute to 100 ml with water.

5.12 Sodium thiosulfate, $Na_2S_2O_3$: crystal.

5.13 Uranium standard stock solution, 1000 microgram/ml. Weigh out 0.1179 g U_3O_8 into a 100 ml beaker and dissolve in 10 ml 1N HNO_3, warming on a hot plate as required. Transfer to a 100 ml volumetric flask with water and dilute to volume.

5.14 Uranium standard solution, 10 micrograms/l. Transfer 5.0 ml of the 1000 micrograms/l uranium solution to a 500 ml volumetric flask and dilute to volume with 0.1N HNO_3.

5.15 Uranium standard solution, 1 microgram/l. Transfer 10.0 ml of the 10 micrograms/l uranium solution to a 100 ml volumetric flask and dilute to volume with 0.1N HNO_3.

5.16 Uranium standard solution, 0.1 microgram/l: Transfer 10.0 ml of the 1.0 micrograms/l uranium solution to a 100 ml volumetric flask and dilute to volume with 0.1N HNO_3.

6. Procedure

6.1 Direct Analysis (Samples greater than 20 micrograms/l).

6.1.1 Transfer two 100 ul aliquots of the filtered sample to each of two gold dishes and evaporate to dryness under heat lamps.

6.1.2 To one of the gold dishes all 100 ul of a uranium standard (0.1 micrograms/ml for samples 20-400 micrograms/l or 1.0 micrograms/ml for samples greater than 400 micrograms/l).

6.1.3 Evaporate to dryness under a heat lamp.

6.1.4 Using a balance sensitive to at least one milligram, weigh out 400 ± 4 mg flux into each of the two gold dishes.

6.1.5 Prepare a blank flux sample by weighing out 400 ± 4 mg flux into a clean gold dish.

6.1.6 Place the three gold dishes into a stainless steel support and place in a preheated muffle furnace at 625° C for 15 minutes.

6.1.7 Remove from furnace and cool in a desiccator for 30 minutes.

6.1.8 Read in a fluorometer as directed in Section 7.0.

6.2 Coprecipitation (Samples less than 20 micrograms/l).

6.2.1 Measure a 1 liter aliquot of filtered water into a 1500 ml beaker.

6.2.2 Acidify with 2 ml 16N HNO_3 (This may be omitted if sample was previously acidified for preservation).

6.2.3 Add 5 ml each of the aluminum nitrate and diammonium hydrogen phosphate solutions and mix.

6.2.4 If sample was chlorinated as in the case of a drinking water sample, add one crystal of sodium thiosulfate and stir.

6.2.5 Heat to near boiling to expel dissolved carbon dioxide gas.

6.2.6 Add 5 drops of phenophthalein indicator and neutralize to the pink end point using 15N NH_4OH.

6.2.7 Lower the heat and digest sample for 30 minutes.

6.2.8 Remove from heat, cool, and settle for one hour.

6.2.9 Decant and filter the clarified supernate through a 47 mm glass fiber filter, transferfering the settled precipitate at the very end.

6.2.10 Wash beaker and filter with small portions of water.

6.2.11 Fold filter into thirds (similar to the folding of a letter) and transfer to a 50 ml screw cap centrifuge tube.

Note: If some of the precipitate remains on the inside edges of the filtering apparatus, gently wipe with the folded filter before transferring to the centrifuge tube.

6.2.12 Add 15 ml 3.5$\underline{M}$ $Mg(NO_3)_2.6H_2O$ to the centrifuge tube to dissolve the aluminum phosphate.

6.2.13 Add 10 ml ethyl acetate, securely cap the tube and mix thoroughly for one minute using a vortex mixer.

6.2.14 Centrifuge at 2000 rpm for 5 minutes.

6.2.15 Using a Pasteur transfer pipette, transfer about 9 ml of the top layer (ethyl acetate) to a 30 ml beaker.

6.2.16 Repeat steps 6.2.12 to 6.2.15 two more times.

6.2.17 Slowly evaporate the combined ethyl acetate fractions to dryness.

6.2.18 Add 1 ml 16$\underline{N}$ HNO_3 and dissolve residue.

6.2.19 Using the same Pasteur pipette, transfer the nitric acid to a 5 ml volumetric flask.

6.2.20 Add 1 ml of water to the beaker, wash down the sides of the beaker using the pipet, and transfer to the 5 ml volumetric flask.

6.2.21 Repeat step 6.2.20 two more times.

6.2.22 Gently mix, cool, dilute to volume with water, and shake thoroughly

6.2.23 Proceed with steps 6.1.1 through 6.1.8, using the 1.0 micrograms/ml uranium standard.

7. Fluorometric Determination

7.1 Place the gold dish containing the sample plus the uranium spike into the fluorometer.

7.2 Following the manufacturer's suggested technique, adjust the voltage to maximize the reading such as full scale deflection.

7.3 Remove the spiked sample, insert the background sample and adjust the null voltage to read zero.

7.4 Repeat steps 7.2 and 7.3 until no more voltage adjustments are required.

7.5 Insert the gold dish containing the sample only and record the output.

8. Calculations

8.1 The results of the analysis are expressed in micrograms per liter and are calculated as follows:

$$\text{Uranium, micrograms/l} = \frac{\left[5 \quad \dfrac{R_s - R_b}{R_{ss} - R_s} \quad x \; a\right]}{b \; x \; V}$$

where:

R_s = Reading of the sample
R_b = Reading of the blank
R_{ss} = Reading of the spiked sample
a = Mass of the uranium spike, micrograms
b = Aliquot size of the concentrate, ml
V = Initial sample size in liters
5 = Volume of the volumetric flask, ml

8.2 In the case where the uranium concentration is greater than 20 micrograms/l and no concentration procedure is performed, the factors "5" and "b" of the above equation are deleted.

9. Precision and Accuracy

9.1 Precision

9.1.1 The single laboratory precision of the method evaluated by replicate analyses of a spiked uranium sample at the 10 micrograms/l concentration. The standard deviation is calculated from the equation:

$$S = \left[\frac{\Sigma(X_i^2) - \dfrac{(\Sigma X_i)^2}{N}}{N - 1}\right]^{1/2}$$

where:

$\Sigma(X_i^2)$ = summation of the squares of the individual results

$(\Sigma X_i)^2$ = square of the summation of the individual results
N = number of results

9.1.2 The coefficient of variation (CV) is calculated from the equation:

$$CV = \frac{100\ S}{X}$$

where:

S = standard deviation from the above equation
X = mean value of the individual results

9.1.3 Using the above equations, the coefficient of variation has been estimated as ± 15%.

9.2 Accuracy or Bias

9.2.1 The single laboratory accuracy of the method was evaluated over the uranium concentration range of 1-10 micrograms/l. The percent accuracy was calculated from the equation:

$$\%\ \text{Accuracy} = \frac{100\ (X_i - X_t)}{X_t}$$

where:

X_i = determined value of individual sample
X_t = known value of the sample

9.2.2 The average percent accuracy, A, is calculated from the equation:

$$A = \frac{\Sigma\%\ \text{Accuracy}}{N}$$

where:

$\Sigma\%$ Accuracy = Summation of the individual accuracy determination
N = number of determinations

9.2.3 The single laboratory evaluation of the average percent accuracy is estimated to be ± 104%.

References

Barker FG et al. 1965. Determination of uranium in natural waters. Geol. Survey Water Supply Paper, 1696-C.

Blanchard R. 1963. Uranium decay series disequilibrium in age determination of marine calcium carbonates. Ph.D. Thesis. Washington University, St. Louis, Missouri.

Edward KW. 1968. Isotopic analysis of uranium in natural waters by alpha spectroscopy. Geological Survey Water Supply Paper. 1696-F.

Grimaldi FS et al. 1954. Collected papers on methods of analysis for uranium and thorium. Geological Survey Bulletin. 1006.